IMPERIAL BUREAU OF ANIMAL NUTRITION

Technical Communication No. 5

Recent Research in Poultry Nutrition

by

A. R. G. Emslie, M.S.A., D.Sc.

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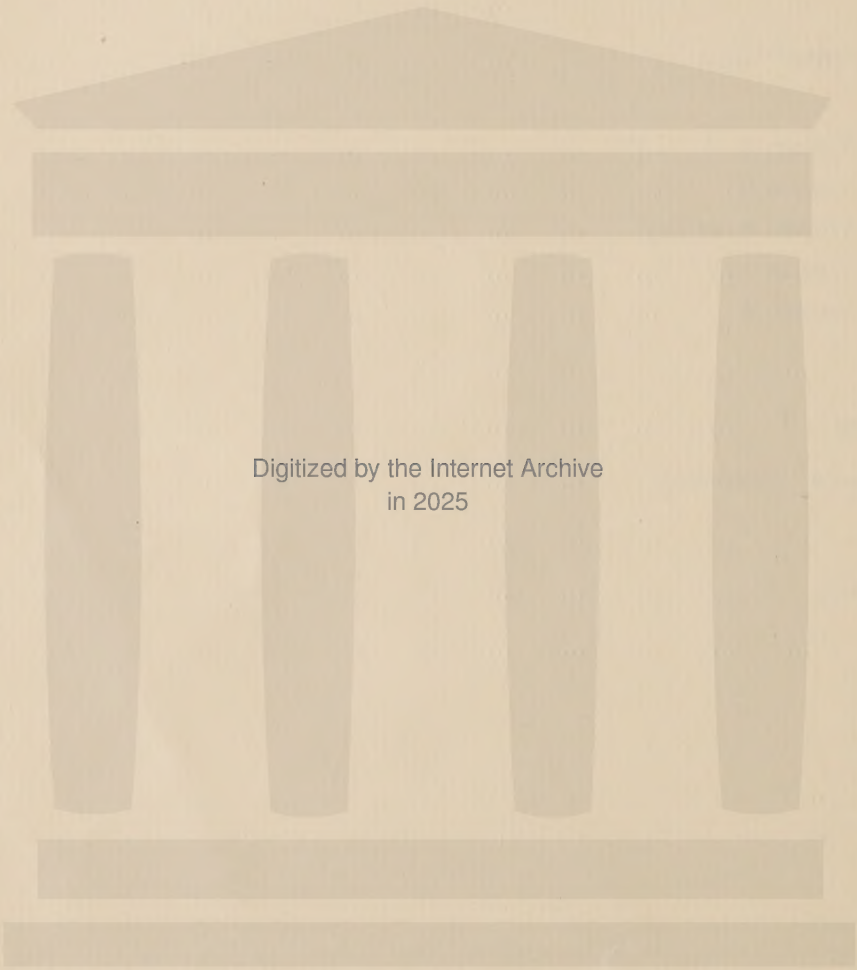

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RECENT RESEARCH IN POULTRY NUTRITION

Introduction.

Poultry husbandry is one of the most important branches of agriculture, and there is still considerable room for its expansion within the Empire. In 1932, for example, the total quantity of whole eggs imported into the United Kingdom was 19,977,000 great hundreds, of which only 5,998,000 great hundreds came from within the Empire. Of this quantity, 3,932,000 came from the Irish Free State; 1,515,000 from Australia; 528,000 from the Union of South Africa; 16,000 from New Zealand; 5,000 from Canada and 2,000 from other Empire countries. [Empire Marketing Board, 1933.]

It used to be considered that little training was necessary for one to become a successful poultry farmer, but now it is becoming more evident that such is by no means the case. The health of the flock can be maintained only by careful and intelligent management, and for this a knowledge of nutrition plays a very large part. When birds have access to good grass runs they may compensate for deficiencies in their ration, but with the advent of battery brooders for chickens, and indoor lofts for hens, it has been found that slight errors in the composition of the rations may cause very serious trouble.

Fowls are becoming increasingly popular as experimental animals, as they are very suitable for certain investigations. This, together with the need for solution of practical problems, has led to a study of their growth under more or less definite conditions, and their requirement for various food substances. Only until fairly recently, however, have any of the poultry research stations specialised in some one phase of poultry nutrition. Reports are published in many journals which not all investigators have the opportunity of seeing, and there is a real need for a review of the literature dealing with different branches of poultry husbandry.

In this monograph, therefore, a few of the salient features of recent research in poultry nutrition are discussed fully. A detailed review of all the experimental work with fowls done during the last few years has not been attempted. The report should be of service to those who are not in close contact with experimental work: however, all the subjects have not been treated "popularly" as such a method often detracts from the value of a review of this type for those most closely interested.

Poultry research presents a very wide field to investigators. Apart from problems connected directly with poultry husbandry itself, there are

many associated with biochemistry, genetics, nutrition, physiology and pathology, the solution of some of which it is hoped will ultimately assist the poultry farmer. One cannot, of course, take the results of laboratory workers, *in toto*, and apply them to fowls kept under commercial conditions, any more than one can argue from farm to laboratory practice. At the same time, it would be of great advantage to the industry if investigators had a knowledge of both poultry husbandry and the higher sciences. All too often are the results of experiments invalidated because certain essential data are not obtained.

The general plan of the report has been to discuss first some of the problems connected with chickens, then with adult fowls and lastly with the incubation of eggs. The practical bearing of experimental results has been borne in mind throughout but has not always been stressed, as it is often difficult to evaluate the significance of certain data from the point of view of the poultry husbandman.

It is outwith the scope of this review to formulate rations for the commercial feeding of poultry during different periods of growth and production, and to offer detailed suggestions as to the types of housing and management to use. Conditions in various parts of the Empire are so dissimilar that one is better advised to consult the publications of the Departments of Agriculture, etc., in the country concerned. In the first section, however, many of the diets have been given in detail, as data obtained under laboratory conditions are usually applicable to investigations throughout the world.

Growth on "Purified" Diets.

Many experiments have been planned to discover the "fundamental" nutritional requirements of chickens by the use of diets, the detailed composition of which is known in a chemical sense. The underlying reason for undertaking such work is that it is thought if investigators know all constituents (inorganic and organic) necessary for the nutrition of fowls, and the effect of varying degrees of deficiency or excess of these, they will be better able to suggest the probable faults of rations in use commercially and the ætiology of nutritional disorders, and to formulate "ideal" rations for different conditions. The work with rats in this regard has progressed further than that with chickens, but the poultry investigator has been unable to employ all the discoveries made with the mammal and to apply them, unmodified, to the bird. Indeed, even within the Family *Aves* one has to be cautious in applying the results to one genus which were obtained with another. For example, while pigeons will grow on a diet of rice, this is not the case with chickens [Drummond, 1916].

Natural foodstuffs contain many little-known food constituents and so to avoid fallacies from the presence of these, numerous attempts have been

made to rear chickens on so-called " synthetic," " simplified " or " purified " diets. It is unnecessary here to review all the literature on the subject but a few of the more successful attempts may be discussed. In the first place, it should be said that one of the chief difficulties encountered in experiments of this type has been the occurrence of " leg weakness." This is a generic term which denotes any condition in which a bird is unable to walk or stand normally, and includes those with symptoms varying from slight staggering to complete paralysis. Specifically, " leg weakness " may be caused by a great many individual and separate factors, and each of these need not give rise to a distinct type of the malady. For example, during prolonged starvation, during an attack of coccidiosis and in vitamin B_1 deficiency, chickens may present very similar outward evidences of " leg weakness." It is necessary to bear in mind in this connection that even when, as judged by photographs, apparently the same type of " leg weakness " has developed on different diets, this need not necessarily mean that the same fault is at work in each case. Something more definite than mere attitude and posture is required for a correct diagnosis : this is discussed more fully in the section dealing with " leg weakness."

Buckner, Nollau and Kastle [1916] found that lysine was necessary for the growth of chickens, as had already been found to be the case for rats. A similar result was obtained by Osborne and Mendel [1916] who were able to rear chickens fairly successfully in the laboratory, except that a number of the birds developed " leg weakness " [Osborne and Mendel, 1918]. Hart, Halpin and Steenbock [1920, 1922] also encountered this latter difficulty. The diet they fed was a modification of one previously used successfully with rats, and was composed of white maize 97, $CaCO_3$ 2, NaCl 1 and skim-milk and grit *ad lib.* The birds died in about six weeks on this diet, and the inorganic-P values (1·07–2·08 mg./100 ml. plasma) were taken by the authors as indicative of the presence of rickets. The addition of 5% of cod-liver oil resulted in better growth, without the same incidence of the leg conditions, and the inorganic-P values rose to 2·50–5·15 mg./100 ml. plasma. A diet composed of dextrin, casein, dried yeast, agar, a salt mixture and saponified cod-liver oil also gave fairly good results when the birds had access to sunlight [Hart, Steenbock and Lepkovsky, 1924]. Hogan, Guerrant and Kempster [1925] used a somewhat similar diet but did not obtain very good growth, and many of the birds developed " leg weakness." That this condition was not the same as that noted by the Wisconsin workers [Hart, *et al.*] was suggested by the fact that it was not prevented by cod-liver oil, but could be prevented by increasing the dried yeast to 10% of the diet, although growth was still subnormal. When polished rice was used in place of the corn starch, and the remainder of the diet kept the same, considerably better growth was obtained : " leg weakness " was reduced to a minimum, and the birds did not have the ruffled appearance noticed previously. Egg yolk improved the corn starch diet, but leg abnormalities still occurred ; the feathers were ruffled and growth was no more rapid, although

somewhat more uniform than on the rice diet. The " leg weakness " could be cured by oral administration of yeast paste or of the B concentrate of Osborne and Wakeman [1919]. The addition of alcohol-ether extracted beef muscle resulted in no improvement.

With their diet of meat residue, peptone, salt mixture, starch, lactose, fibre, lard and extracts containing vitamins A and B, Emmett and Peacock [1923] obtained quite good growth and no leg abnormalities. Hogan and Shrewsbury [1930–31] reported on experiments planned to elucidate the cause of the " leg weakness " they had encountered in their laboratory. The basal diet they used was composed of casein (leached with acidified water) 35, maize starch 28, dried yeast 15, lard 10, cod-liver oil 5, cellulose 3 and a salt mixture 4 parts. The chief trouble met with was a condition they thought similar to polyneuritis. Various sources and amounts of vitamin B-rich substances were tried, for as already mentioned, yeast paste had cured the condition in previous experiments. With large amounts of the Osborne-Wakeman vitamin B concentrate in the diet, there was good growth up to the third week, after which a number of the chickens collapsed with neuritic symptoms, such as spastic leg muscles, sometimes tremors and mild head retraction. Skimmilk *ad lib.* improved the growth but did not reduce the incidence of " leg weakness." Neither did 15% of yeast, dried at low temperatures, nor the equivalent amount of fresh yeast, when mixed in the ration. Oral administration of yeast paste was effective, and increasing the dried yeast in the diet to 20 or 30% levels proportionately lessened the incidence of the leg disorders and improved the growth. Even with 40% of yeast, however, while the results on the whole were good, there were a few cases of " leg weakness," but these recovered without treatment. An aqueous extract of yeast, concentrated and dried, gave good results when fed at a level of 20%. This was considered on the borderline of the requirement, for occasional cases showing the leg condition still appeared ; these were cured by further treatment with yeast, *per os.* The authors concluded that, to be successful, " simplified " diets must be fortified with approximately 40% of dried yeast, or 20% of a water extract (dry basis), although the diet could then hardly be called a " synthetic " one.[1]

The beneficial effects of replacing the starch or dextrin of this type of diet for chicks with white rice were also noted by McFarlane, Graham and Hall [1931] when they were studying the influence of various protein supplements. They used a modification of the diet employed by Hart, Steenbock and Lepkovsky [1924]. The protein supplements were ether-extracted, and supplied 18% crude protein. The other ingredients were yeast extract [method of Hartwell, 1922] 10% ; butter oil 10% ; saponified cod-liver oil 2% ; paper 10% ; fish meal ash to 8·9% total ash, and dextrin to 100%.

[1] Hogan and Boucher [1933] have recently reported some success in rearing birds to the second generation by the use of " purified " diets supplemented with extracts of yeast, liver, etc.

The chicks received U.V. Irradiation. The best growth was obtained with the diet containing buttermilk powder, but " leg weakness " occurred after 9–18 days, with paralysis at about 20 days in both legs and wings. The chicks were dirty in appearance owing to the wet nature of the excreta. There appeared to be some anæmia and a low blood volume. Of the groups of chickens, that receiving fish meal as the supplement grew most rapidly at first but more slowly after 25 days. " Leg weakness " was common and the anæmia somewhat worse. · Increased amounts of yeast extract did not improve either of these two diets. With cod-liver meal, growth was quite good up to 18 days, when considerable diarrhœa occurred. This was improved, however, by the inclusion of 5% charcoal in the diet. The hæmoglobin was the highest of the groups of chickens tested.

In the buttermilk powder diet, when the yeast extract was replaced by untreated brewers' yeast, growth was improved, but the " leg weakness " was worse, and not cured by increasing the yeast to 20%. That this was not a rachitic condition was indicated by the high femur and tibia ash percentages of 55 and 53 respectively. When tomato juice, 20 ml. daily, replaced the yeast extract there was no improvement in growth. On a diet containing gelatin and brewers' yeast, the chicks failed to grow and developed cannibalistic tendencies. Very little improvement was noted when the basal diet was changed to include dried brewers' yeast and cod-liver oil. With buttermilk as the protein supplement there was no significant change in the incidence of " leg weakness " when the ratio buttermilk powder protein : yeast protein was 1 : 1 or 1 : 2.

The basal ration was then changed to one having the following composition : marmite 15, cod-liver oil 3, iodized salt (later discontinued) 1, bone meal to 4·36% total ash, and white rice to 100. U.V. Irradiation was given for 10 minutes each day. Growth in this series was considerably better than in the previous experiments, and " leg weakness " did not occur. It will be noticed that the most significant changes in the basal diet were the use of white rice in place of dextrin, and the lowering of the total ash percentage to one-half of what it was in the former diet. Growth was equally good with either buttermilk powder or casein, but when dried brewers' yeast was used in place of marmite in the diet with casein, some birds appeared " polyneuritic " at 4–5 weeks, although they recovered later. Fish meal and meat meal gave similar results, but the birds were " bleeders," some dying from hæmorrhage when the identification bands were transferred from the legs to the wings. The chicks receiving cod-liver meal did not grow so well, weighing only 375 g. at 8 weeks compared with the buttermilk powder birds which weighed 500 g. at the same age. Normal growth resulted only when white rice with either marmite or dried brewers' yeast were included in the basal diets. The authors concluded that either some vegetable protein was necessary (the white rice supplied approximately 2·6% protein) or else the rice contained some other factor necessary for growth. A diet which approached the " purified " type, and which resulted

in normal growth consisted of :—casein 12·2, marmite 15, cod-liver oil 3, salt mixture 4·4 and white rice 61.

Further experiments [Graham, Pettit, Sykes and Howell, 1934] showed that extraction of the white rice with solvents such as alcohol, ether and acetone did not markedly affect its growth-promoting properties. The addition of wheat germ to a diet containing maize starch in place of the rice gave good results. Different extracts of wheat germ middlings varied in their value : acid extracts were on the whole better than alkaline extracts. The " slipped tendon " condition was observed : it did not occur with the acid extracts nor with the residues after alkaline extraction, but was common with the alkaline extracts and less so with the residues after acid extraction. A diet which allowed of normal growth up to 12 weeks of age consisted of :— " technical " casein 20, yeast 15, wheat germ 10, salt mixture 4, cod-liver oil 5 and maize starch 46 parts.

Summary.—Work on simplified diets has not yet progressed sufficiently far for one to be able to tabulate all the nutritional factors required by chickens. It would appear that 20% or somewhat less of casein supplies a sufficient quantity and quality of protein : it is possible, however, that the protein of dried yeast is of significant importance. Carbohydrates may be fed in the form of starch or dextrin, up to about 50% of the diet. The fat requirement has not been worked out, but it appears to be very low : nevertheless, the increase in the fat content of the diet may be one reason why Graham, *et al.* [1934] obtained better results with 5% than with 3% of cod-liver oil.

When vitamin D is supplied the mineral content of the diet may be varied considerably, but 4% of the mineral mixture of Hart, Halpin and Steenbock [1920] appears to give quite good results.

Of the vitamins, those of the B group would appear to be the most difficult to supply in a reasonably purified form. Their relative deficiency may result in lack of maintenance or growth, or in the production of some types of " leg weakness." Of the latter there appear to be at least three forms encountered in these experiments with " purified " diets—as judged by the effect of curative agencies. There are the polyneuritic type ; that observed by Hogan and Shrewsbury ; and that of McFarlane, *et al.* which was prevented by replacing dextrin by white rice. The " slipped tendon " condition which Graham, *et al.* found when wheat germ middlings was extracted with acid solvents, has yet to be shown to be due to a vitamin deficiency. A fuller discussion of the vitamins and of forms of " leg weakness " follows.

While this work may have little immediate practical bearing, it is probably of more than mere scientific interest. Its successful continuance should mean that it will be possible to study in the laboratory an increasing number of nutritional disturbances affecting fowls kept commercially. With a knowledge of the ætiology, curative and preventive measures can be instituted to suit different local conditions.

The Vitamins.

The chicken's requirement for some of the vitamins has received considerable attention, but more from the practical rather than the scientific standpoint. Laboratory experiments, however, have been carried out. Poultry investigators realise the fact that results obtained with one type of animal may not be applicable to another, and that much of the work done with rats will need to be repeated with fowls before its significance for poultry can be understood.

Vitamin A.

Many investigators have shown that chickens require vitamin A. In their study Elvehjem and Neu [1932] have, for the most part, confirmed the observations of previous experimenters with regard to the symptoms of deficiency of this vitamin. On one diet they used, consisting of white maize, $CaCO_3$, NaCl, skimmilk *ad lib.*, together with U.V. Irradiation, there was cessation of growth before the appearance of the typical symptoms. With a diet of white maize 58, wheat middlings 25, crude casein 12, NaCl 1, precipitated $CaCO_3$ 1, precipitated phosphate 1, and dried yeast 2, together with U.V. Irradiation for 20 minutes three times a week, they obtained good growth up to three weeks of age, at which time certain symptoms developed, and the birds ceased growing or even declined in weight. A staggering gait, incoordination of movements, drowsiness and disinclination to move, with the assumption of a crouching posture were the chief features. In advanced cases the birds lay on one side with their heads forward, their feathers ruffled and their eyes inflamed. There was no loss of appetite in the earlier stages of the disease. Most of the chickens died before the fifth week, but the addition of 2% of cod-liver oil to the ration permitted normal growth to proceed. *Post mortem* examination showed in the majority of cases the deposit of urates on the visceral organs which is considered typical of vitamin A deficiency in chickens. They found the blood uric acid of normal birds to be 5 mg. per 100 ml. blood, with a tendency to somewhat higher levels with increasing age : there were almost invariably considerably higher levels in birds suffering from vitamin A deficiency, values as high as 44 mg. per 100 ml. blood being observed. This was not correlated with age, but was related, in general, with the severity of the muscle incoordination. Histological examination of the kidney revealed slight nephrosis. The authors suggested that the high blood uric acid was not due to disturbed uric acid metabolism but to kidney dysfunction, while the incoordination was probably due to nerve changes.

That carotene can supply the vitamin A requirements of fowls as in other animals is suggested indirectly by the benefits obtained from the use of yellow maize. Hauge, Carrick and Prange [1926–27] found that a ration containing 25% of yellow maize was just within the limits of safety, if no other source of vitamin A were given, for rearing chicks to maturity. Capper,

McKibbin and Prentice [1931], using just under 16% of yellow maize in their rations, observed the symptoms of vitamin A deficiency after about 16 weeks. The livers of these birds gave negative results when tested colorimetrically. The addition of carotene (0·5 mg. per bird per day) met the vitamin A requirements.

Kline, Schultze and Hart [1932, 1] using the same diet as Elvehjem and Neu [1932], found that for prophylactic purposes 0·03 mg. daily of carotene was not quite sufficient for normal growth, especially when the birds were about seven weeks old. They suggested that the requirement was related to the weight of the bird. In their curative tests 0·05 mg. carotene permitted a resumption of growth, but this amount appeared to be insufficient at the eighth week, when the birds weighed 400 g. They found xanthophyll to have no effect in preventing or curing the avitaminosis, even in doses of 0·25 mg. daily. At this level there was only a slight increase in yellow pigmentation of the shanks: in their earlier work Palmer and Kempster [1919] had found a definite increase in shank colour when they fed larger amounts of xanthophyll.

With regard to other sources of vitamin A, cod-liver oil is probably the commonest employed for prophylactic purposes. Gutteridge [1931–32] found that even at a 4% level pilchard oil was not so effective as cod-liver oil, although its vitamin D content was about the same. Similarly Cruickshank, Hart and Halpin [1927–28] found that cod-liver meal contained vitamin D but that 10% in the ration was an ineffective source of vitamin A. When birds have access to good grass runs or when there is sufficient alfalfa leaf meal or similar product in the ration, the likelihood of there being vitamin A deficiency is small. Hewang and Titus [1932], using mortality of the chickens as the criterion, found alfalfa leaf meal superior to alfalfa meal. At a level of 10% in a ration otherwise deficient in the vitamin, the former was not entirely satisfactory. Of the other foodstuffs tested, 1·5% of cod-liver oil was better than fresh alfalfa (approximately 7% of the ration, dry matter basis) and this in turn better than yellow carrots. Payne and Hughes [1933] found 10% of alfalfa leaf meal supplied sufficient vitamin A for laying hens.

Summary.—The clinical appearance of chickens suffering from vitamin A deficiency is not very specific. The feathers are ruffled and "staring," and the birds seem hunched up. The high blood-uric acid is an aid in the diagnosis, but under practical conditions it is generally sufficient to take as a criterion the probable vitamin content of the ration. When yellow maize is over 25%, when there is a good deal of alfalfa leaf meal (over 10% in an otherwise vitamin A-poor ration) or at least 1·5% of cod-liver oil a deficiency of this vitamin is improbable. This is also the case when the birds have access to a good grass range. Under the conditions now prevailing in most countries, vitamin A deficiency has ceased to be a serious problem, at least for growing chickens.

Vitamin D.

In the majority of reports of poultry experiments the term rickets—when used at all in a specific sense—is intended to indicate a disease in which the ash percentage of the long bones is subnormal, and any condition in which the ash percentage is low is usually regarded as rickets and, therefore, as preventable by vitamin D. Unfortunately it is seldom realised that the bone ash method may be only a rough index, and that it may be by no means a specific test for rickets. Non-realisation of this is all the more regrettable in view of the large number of experiments which have been carried out for the purpose of studying some phase of the vitamin D requirement of chickens.

For the purpose of this review, however, owing to the almost exclusive use already made of the estimation of the bone ash percentage in a diagnosis of " rickets," and to the paucity of histological descriptions of the conditions actually obtained, the observations in the literature are discussed from the standpoint that vitamin D inadequacy results in subnormal ash percentages (below 40%, fat-free basis) of the long bones. This appears to be the only common ground for discussion of the data to hand, but one is thereby not committed to the view that the conditions observed are all rickets.

The details of the symptoms and of the pathology of vitamin D deficiency of chickens seem none too certain at present, partly owing to the omission of due consideration of the effect of other factors which may be either lacking or in excess. Investigators have so far been unable to rear chickens successfully to maturity without supplying some source of vitamin D, and it may be taken as a general rule that a prolonged deficiency of the vitamin will result in a disturbed mineral metabolism in growing chickens, regardless of the amounts or ratio of calcium and phosphorus in the diet.

The chief symptoms reported are a staggering gait, reluctance to stand or move about, bent legs, beading or " waving " of the ribs, crooked breast bones, soft beaks and drowsiness. Loss of appetite is said to occur only in severe cases, when death finally intervenes. A lowering of the serum-Ca and/or blood inorganic-P is the general finding in young birds, but this may not occur in adults. While investigators are agreed that the ash percentages of both femora and tibiæ are low, all do not follow a standard method in their analysis and it is difficult to compare their results. Some calculate on the basis of the " green " bone, others of the dried unextracted bone but the majority take the dried extracted bone. Methods of extraction differ. This has been discussed by St. John, Kempf and Bond [1933]. They found that on the basis of the dried extracted bone, the average ash percentages of the tibiæ of 8-week old chickens on a " rachitogenic " diet were as follows :—tibia 41, shaft 50, distal cartilage 26·4, proximal cartilage 11·2. The same diet supplemented with vitamin D gave the following results :—tibia 52·1, tibia shaft 59·0, distal cartilage 38·7, proximal cartilage 16·5. The method they recommend is briefly as follows. The flesh, tendons and both cartilage caps are removed from the tibiæ, which are then dried for at least 24 hours

at 65° C. The bones are then extracted for 48 hours with 95% alcohol. After being dried at 100° C. to constant weight, ashing is carried out in a muffle furance at 650° C. for 3 hours, or until a white ash is obtained. The percentage of ash is calculated on the dry, fat-free bone. No difference was observed between the tibiæ of the same chicken.

X-ray examination is commonly used to show the condition of the ribs or the degree of calcification of the long bones. The shadow of the epiphyses is said to be very similar to that of mammals with rickets.

The histological appearance of the bones at their articulations is generally considered not to be identical with that found in rickets in rats. Thus Pappenheimer and Dunn [1925] were unconvinced that the condition they observed was true rickets—there are so many different types of " leg weakness " and their conclusion seems warranted. Hart, Scott, Kline and Halpin [1929–30], in discussing the " line test " as applied to the tibiæ, pointed out that in both the rat and the chicken " there is a wide uncalcified area ; however, on healing the calcification in the case of the rat begins at the middle or outer border of the uncalcified metaphysis, and with the silver nitrate test this is shown as a line of deposited calcium salts. In the case of the chick, the healing begins at the border of the metaphysis already calcified, and gradually pushes forward toward the end of the bone. In healing rickets in the chick there is no definite line of calcification shown by the silver nitrate test, but a gradual narrowing of the uncalcified area in the metaphysis through a deposition of lime salts immediately contiguous to the area already calcified." The fullest histological description of " rickets " in the fowl is probably that of Nonidez [1928]. In his opinion, bone formation of the newly hatched chick is considerably behind that of the new born mammal (the rat), and if vitamin D be withheld from hatching onwards, the resulting histological appearance is one of osteoporosis. Only when the birds were allowed to grow normally for five weeks and the vitamin then discontinued did lesions similar to mammalian rickets develop. Baby chicks were fed a grain and cereal ration, together with semisolid buttermilk. For the first five weeks they were allowed in the sun, but were thereafter confined to the brooder house behind glass. " Leg weakness " occurred during the succeeding four to five weeks. Microscopic examination of the long bones showed " excess of osteoid in the spongiosa ; active production of periosteal osteoid ; increase in thickness of the osteoplastic layer throughout the bone of the rachitic band ; abundance of osteoclasts ; absence of calcification in the zone of provisional calcification."

Vitamin D deficiency is associated with an increase activity of the bone phosphatase. The values observed by Hall and King [1930–31] were twice that of the normal, although changes in the kidney and intestinal phosphatase were insignificant. Plasma phosphatase values are also higher than normal.

Sheehy and Sheil [1932] have shown that early vitamin D deficiency, even if rectified later, may result in distortion and decrease in the size of the thoracic cavity, and decrease in the size of the internal organs of chickens.

Cod-liver oil is the commonest substance used to supply vitamin D in the ration of confined birds. A difference in the potency of various grades of cod-liver oil has often been shown by tests with rats, and Heuser and Norris [1926–27] found that for chickens this was true also. Brown oil was as effective as American refined and tested oil, and somewhat superior to the refined but non-tested samples. In general the American oils were better than the Norwegian products. That Newfoundland oils are more potent than those of Norwegian origin as shown by Drummond and Hilditch [1930] would seem important from an Empire standpoint. Parkhurst [1930] and Sheehy [1930] stated that from their experiments some oils were ineffective at a 2% level, while 0·5% of other samples gave good results. Kitto [1932–33] obtained no benefit from the use of cod-liver oil in N. Zealand, but full details of his experiments were not reported. Holmes, Moore, Bennett and Pigott [1929–30] found that red oil of high acidity might be detrimental.

The minimum effective dose naturally varies with the type of ration used. Dunn [1924] and Plimmer, Rosedale and Raymond [1927] were among those who found that 0·5% of cod-liver oil in the ration gave as good results as higher amounts. McFarlane, Graham and Richardson [1931], however, suggested that the fish meal used by Plimmer and his associates might have been a source of vitamin D: they found that under laboratory conditions, with meat meal as the protein supplement, the requirement was in excess of 1% of the cod-liver oil they used; with fat-extracted fish meal, 3% was not the minimum. Unextracted fish meal, on the other hand, was not improved by the addition of cod-liver oil. The percentage of crude protein was the same in all the experiments. Under practical conditions it is probable that 0·5% of a good grade oil is sufficient, but many consider 1% to be advisable. The question of whether or not one should pay the higher price for tested oils would seem to depend upon the poultryman himself. Probably the "unrefined" and "cattle" grades sold by reputable firms, more especially possibly if the oils are of Newfoundland origin, will give better results per unit price than the highly refined oils [but *vide supra* Parkhurst, 1930; and Sheehy, 1930].

Dunn [1924] found that when cod-liver oil was stored in starch it decreased rapidly in antirachitic potency: when the oil was mixed in the mash and stored in galvanised cans, Hart, Steenbock and Lepkovsky [1925] observed no deterioration for at least six months. Holmes and his collaborators [1930–31] suggested that with an oil of low acidity and high potency, storage of the mixed mash for a year was not detrimental, and Sheehy [1931–32] obtained good results after 16 months of storage.

Some other fish oils which have been found effective in preventing "leg weakness," or allowing of normal growth in chickens are as follows: 2% salmon oil extracted from cannery refuse, by Davis and Beach [1927–28]; 0·5% and 1% of sardine oil by Mussehl and Ackerson [1933] and Gutteridge [1931–32] respectively; 1% pilchard oil by Gutteridge [1931–32]. Asmundson,

Allardyce and Biely [1928–29] found that 2% of pilchard oil was not quite so effective as 2% of cod-liver oil, and dog fish oil was decidedly inferior. The potency of different samples of the latter varied considerably.

Munro and Rae [1929–30] found that oat oil, even when irradiated, possessed no antirachitic properties for chickens.

Properly irradiated ergosterol is the most active antirachitic substance known, the preparation known as calciferol having 40,000,000 international units per gram. Rats are used in the routine method of assay for vitamin D, however, and there is some question as to whether work done on rats is applicable to chickens. It has, indeed, been shown by many investigators that solutions of irradiated ergosterol having the same vitamin D potency for rats as samples of cod-liver oil are not so effective as the latter in preventing "leg weakness" in chickens receiving an otherwise "rachitogenic" diet. There is some doubt as to what constitutes the exact difference. Massengale and Nussmeier [1930] concluded that 200 times more vitamin D was required when supplied as irradiated ergosterol than that present in 2% of cod-liver oil, and Steenbock, Kletzien and Halpin [1932] found that it took 40–120 times as many rat equivalents of the sterol to give results similar to 1% of cod-liver oil. Mussehl and Ackerson [1929–30] found that between 200 to 5,000 units per 100 g. of the ration were ineffective, but King, Hull and Hall [1933] concluded that if the ergosterol were given by mouth and not mixed in the ration, the vitamin D of the sterol was as effective as that of cod-liver oil. The latter workers stressed the importance of having other constituents of the ration, such as Ca and P present in the right amounts and proportions.

In their series of experiments to discover the ultimate fate of vitamin D Klein and Russell [1931] found that, by the rat assay method, about 26% of the antirachitic factor fed as irradiated ergosterol, and about 43% when fed as cod-liver oil, could be recovered from the excreta of chickens. The remainder of the vitamin D which was fed could not be recovered from the bodies of the birds. There was no significant difference in the effect on the bones, as determined by the ash percentages, in comparative tests where the ergosterol solution and the cod-liver oil were given by capsule and injected intraperitoneally; but the bones of the birds receiving cod-liver oil were better calcified than the bones of the birds receiving the ergosterol. These and other investigators [Russell and Klein, 1930–31; King and Hall, 1930–31; Hunter, Dutcher and Knandel, 1932] have shown that this difference in the antirachitic effect of cod-liver oil and irradiated ergosterol preparations cannot be explained on the basis of a vitamin A and D imbalance, nor on one of a "sparing" effect of vitamin A of cod-liver oil which is lacking in the ergosterol solutions. The addition of vitamin A or carotene to the ergosterol does not enhance its antirachitic effect for chickens. That there should be this apparent difference between the potency of the two substances for chickens was commented on by Hess and Supplee [1930] as being all the more remarkable because of the effectiveness of ultraviolet irradiation for

poultry. Steenbock and his fellow workers [1932], indeed, were among those who suggested that possibly the vitamin D for rats is not the same as that for chickens.

This is quite an important question from the practical standpoint, for many commercial poultry feed manufacturers use irradiated ergosterol in their mash mixtures. So far as it appears from the literature, there has been no detailed report of ill effects of the nature of a high incidence of the rachitic type of " leg weakness " following the use of such mashes. Probably the majority of the firms test their products in some way occasionally, and no doubt if the rations were otherwise rachitogenic in character and the chickens received no sunshine, the probable ineffectiveness of the ergosterol in other than large doses (rat unit basis) would have been noticed. Why laboratory workers have observed the discrepancy may possibly be understood if one considers their more carefully controlled conditions, and the greater severity of their tests. While the introduction of battery brooding by poultrymen has lessened the gap between " laboratory " and " commercial " conditions of rearing chickens, perhaps the popularity of fish meal, many samples of which are rich in vitamin D, should be taken into account in explaining the discrepancy.

Some study has been made of the effects on chickens of large doses of irradiated ergosterol, but as with the protective dose there is some difference of opinion as to what amount must be given to prove toxic. Kreitmair and Moll [1928] concluded that poultry were very resistant to an excess of vitamin D. Massengale and Nussmeier [1930] found that 2,000 times the antirachitic dose was not toxic when the Ca : P ratio of the diet was low. On a diet containing 6% $CaCO_3$ [Ca : P = 8 : 1] there was a slight rise in serum-Ca but otherwise the birds appeared quite normal. Hall and King [1930–31], on the other hand, using a diet having a Ca : P ratio of 1·8 : 1, observed a slight rise in serum-Ca and fall in serum inorganic-P after about the sixth week, and growth ceased after about the ninth week, when the birds began to lose weight. The bones were found to be decalcified and the bone phosphatase was lower than normal. No calcification of the soft tissues was seen on microscopical examination. Taylor and his collaborators [1931] found little difference between control chickens and those receiving 25,000 times the probable antirachitic dose : with larger doses there was a slight rise in serum-Ca. They estimated the lethal dose to be over a total of 800,000 rat units. Their chickens were 5 months old, and were fed on a mixture of yellow maize 1, wheat 1, and oats 2 parts.

In a later paper King and Hall [1931] confirmed their previous results but found, in addition, that daily oral doses of 3–6 ml. of a viosterol preparation (which was 1,500 times as potent as " standard " cod-liver oil for rats) to 5 month old pullets resulted in heavy deposits of Ca in the secretory tubules of the kidney. Bone ash percentages were normal although there appeared to be a low amount of calcareous deposit within the matrix of the trabeculæ of the femur. Bone phosphatase fell to as low as

0·08 units (normal value 3·47 units). Parathormone injection (5–10 units daily) produced no noticeable ill effects, except that "histological examination of the femurs showed progressive fibrosis of the bony trabeculæ without evident deposition of lime salts in the hyperplastic tissue."

The antirachitic value of irradiated yeast has also been compared for rats and chickens. Mussehl and Ackerson [1929–30] reported that when it was fed in doses equivalent to 10 or 30 times the rat potency of standard cod-liver oil, it was ineffective. Steenbock and his collaborators [1932] found that amounts equivalent to 7·5 to 60 times the rat potency of standard cod-liver oil gave results similar to 1% of the cod-liver oil they used.

Opinion differs with regard to the antirachitic potency of other feeding stuffs, partly due to the types of rations used, but probably more especially to the difference in the process of manufacture and the source of material. Bethke, Zinzalian, Kennard and Sassaman [1928] found cod-liver meal, even when irradiated, had no appreciable antirachitic value; Cruickshank, Hart and Halpin [1927–28] found 4% of cod-liver meal in the ration was protective. Fishmeal was found by Holmes, Pigott and Menard [1930–31] to possess no significant amount of vitamin D, but McFarlane, Graham and Richardson [1931] found that when fishmeal supplied 18% crude protein in the ration, the addition of cod-liver oil was unnecessary.

Poultry are quite sensitive to ultra violet irradiation. In laboratory experiments it is common to expose the birds for periods of 10 minutes daily, or 20 minutes three times a week, using the quartz mercury vapour lamp. In their experiments, Russell, Massengale and Howard [1928] found that the effect of a single exposure for 45 minutes filtered (15 minutes direct) radiation at 3 feet with this type of lamp gave a duration effect of one week, while the effect of 90–270 minutes exposure lasted two weeks, as judged by the ash percentages of the bones. A one day exposure to sunshine behind Cel-O-Glas gave a three days duration effect [Russell and Howard, 1931]. This type of glass substitute was still effective from February on through the summer after 13·5 months of weathering [Russell and Howard, 1928–29]. Mayerson and Laurens [1932] found that effective daily exposures to direct sunshine in New Orleans (latitude about 30° N.) were as follows. January, 5 minutes; February and March, 3–5 minutes; April to June, 2–3 minutes; July to September, 2 minutes; October, 2–3 minutes; November and December, 5 minutes. Effective daily exposures to skyshine were as follows. January and February, 35 minutes; March to June, 19–19 minutes; July to October, 6–12 minutes; November and December, 35 minutes. Vita glass and glass substitutes (Corex D and Cel-O-Glas) were effective in correspondingly longer exposures depending on their transmission of the shorter wave lengths. The minimum amount of solar energy of wave lengths shorter than 313 μμ necessary to promote normal growth and calcification of chickens on a rachitogenic diet was about 0·69 mg. cal/sq. cm. (48·3 μw) while with skyshine the amount necessary was about 1·34 mg. cal/sq. cm. (99·8 μw).

Mussehl and Ackerson [1930–31, 1] found 20 minutes daily exposure quite effective when using a " Sunlight " or S.1 type of lamp, which was a combination of tungsten filament, tungsten electrodes and mercury arc. Miller, Bearse and Cushing [1933] found the 60 watt CX Mazda socket lamp useful when the exposures were sufficiently long [7 hours a day]. Gutteridge [1931–32] failed to get good results with a socket type of " Sunlight " lamp, but he did not mention the length of the exposures.

Summary.—Without differentiating between rickets and other bone abnormalities which are prevented by vitamin D, it may be said that the general experience of investigators is that an inadequate supply of vitamin D to growing chickens will result in certain symptoms of malnutrition. Chief of these is the flexible state of the limb and breast bones and beaks. The limb and breast bones often become crooked. *Post mortem* examination will often, but not invariably, reveal a beaded condition of the ribs and enlargement of the parathyroids. The ash percentage of the leg bones is subnormal, and bone and plasma phosphatase values are high. Curative measures are generally very effective if commenced soon enough.

Prevention under commercial conditions can be done most simply, as in the case of chickens in battery brooders where it is inconvenient to allow the birds sunshine, by feeding 0·5–1% of a good grade of cod-liver oil. The unrefined grades sold by reliable firms will probably be found sufficiently antirachitic ; some authors, however, advise the use of tested grades as they consider the extra cost fully justified [*vide supra*]. When the chickens are exposed to direct sunlight, or to that transmitted by reliable glass substitutes, it is probable that only when the hours of sunlight are very short will it be necessary to add cod-liver oil to the ration. It should be remembered, however, that when the windows of the brooder house are of ordinary glass, " rickets " may develop even when the chickens have access to outside runs, as on certain types of rations it has been observed that all birds may not take advantage of the direct sunshine. The minimum requirements of vitamin D seem linked with the optimum amounts and the " availability " of Ca and P in the ration, so these should be considered. In regard to the use of irradiated ergosterol it would seem preferable to withhold definite judgment until further experiments have been carried out to study its relationship to the antirachitic factor for poultry. The use of the term " rickets " has been used too loosely in experiments with chickens for there to be sufficient proof as yet that investigators have been comparing only the *antirachitic* effect of cod-liver oil and irradiated ergosterol [*c.f.* Branion, 1933, 2].

Vitamin B.

It is not very clear at present which factors in the vitamin B complex are essential for the normal health of chickens. When Eijkman in 1897 showed that fowls fed on polished rice developed polyneuritis, it was taken

for granted that vitamin B was necessary for poultry, and when it was shown that B_1 was the important factor in this regard, it was assumed that chickens required that part of the vitamin B complex. Much of the earlier work, however, was done on undifferentiated vitamin B, and in view of the later findings is not sufficiently specific to warrant full discussion here.

The B_1 factor is generally considered to be relatively heat-labile, and to be destroyed by autoclaving at 120° C. for 6 hours. On a diet deficient in B_1 chickens cease to grow after a short time, become unsteady on their legs and may lie on one side, or " squat " with the neck bent backwards, similar to the usual opisthotonic position commonly noted with pigeons used in vitamin B_1 assay. Chickens and older birds may exhibit this condition, but probably more frequently they lie on one side with their legs sprawled out under them. From the work of the Oxford investigators [*vide* Kinnersley and Peters, 1929] on B_1 deficiency in pigeons, it would appear that there is a decreased O_2 uptake by, and an increased lactic acid content of the brain. Administration of torulin, a B_1 concentrate, causes a return of the O_2 uptake and decrease of the lactic acid content of the brain to normal. This also occurs when torulin is added to excised avitaminosis B_1 brain preparations.

Kline, *et al.* [1932, 2] and Elvehjem, *et al.* [1932, 2] described experiments with chickens fed the following diet : yellow maize 58, middlings 25, casein 12, NaCl 1, $CaCO_3$ 1 and cod-liver oil 2 parts. The birds grew to a weight of 300 g. in six weeks. When the diet was autoclaved at 120° and 15 lbs. pressure for 5 hours (the $CaCO_3$ and cod-liver oil being added after autoclaving), polyneuritis developed in 7–10 days. The birds were extremely emaciated, showed general lack of muscular coordination and sense of balance, and opisthotonus usually followed. The syndrome could be cured by yeast administration and prevented by including 4% of air-dried yeast in the diet. The growth, however, was possibly not quite so good as with the unautoclaved diet. When 32% of the latter was added to a modification of the autoclaved diet, good growth occurred, showing that the unautoclaved diet was quite rich in vitamin B_1.

Plimmer's work on the B_1 content of foodstuffs has already been reviewed [Med. Res. Council, 1932], and need not be discussed here. Green foods and germinated grains are rich in B_1 and lettuce has been advocated as a cure for the type of paralysis resulting from B_1 deficiency. Birds receiving a mixed cereal diet will normally obtain sufficient of the vitamin, especially if they have access to good range. Under intensive conditions, however, it may be advisable to give a B_1 supplement. Yeast is becoming more common now as an ingredient for poultry mashes. Hamilton, Card and Kick [1926–27] and Bethke and Kennard [1927–28] obtained no appreciably better results with the addition of yeast when over 60% of the ration consisted of mixed cereals ; had better results been obtained [Mussehl and Ackerson, 1930–31, 2] it would not have been clearly a vitamin B_1 effect. The suggestion made by Blount [1932] that birds may suffer from an excess of vitamin B appears to require considerably more substantiation. As was mentioned in

the discussion on "purified" diets, the inclusion of 40% of yeast in the diet did not appear detrimental to growing chicks.

Autoclaving at 120° C. with 15 lbs. pressure for 5 hours destroys vitamin B_1 only slightly, and this is the common method of treating yeast to obtain B_1-poor and B_2-rich material. The Wisconsin investigators [Kline, *et al.*, 1932, 2; Elvehjem, *et al.*, 1932, 2] pointed out that some of the contradictory results of vitamin B studies have been due to the different methods of extraction used in obtaining concentrates of the different factors. These workers were able to develop a pellagra-like condition in chickens by dry heating the diet, already mentioned in their B_1 studies, at 100° C. for 144 hours. This treatment appeared to destroy B_2 without appreciably affecting B_1. Ringrose, Norris and Heuser [1930–31] had previously described the "pellagra" lesions thus: "These lesions developed at the eyes, at the mouth corners and on the feet. The margins of the eyelids became granular and contracted so that vision was restricted. A viscous exudate was produced which frequently caused the eyelids to stick firmly together. Crusty scabs appeared at the margins of the skin around the nostrils and underneath the lower mandible. The outer layers of the skin on the bottoms of the feet and between the toes peeled off. Afterwards, small cracks and fissures appeared at these points. These enlarged and deepened so that chicks affected were sensitive to walking. In the meantime, the remaining skin layers thickened and cornified and in some cases wart-like protuberances developed on the balls of the feet. Feathering was retarded and the feathers produced were rough and staring . . . in a group of chicks 18 weeks of age . . . complete loss of feathers occurred in limited areas on the head and neck. *Post mortem* examination showed the presence of a pus-like substance in the mouth and an opaque, grayish-white exudate in the proventriculus. Eroded lesions were found in the gizzard and the duodenum usually showed severe enteritis. . . . The liver frequently possessed an abnormal colour, varying from a faint yellow to a deep dirty yellow . . . spleen small and atrophic . . . kidneys . . . appeared grayish-white or inflamed and hæmorrhagic."

At both Ithaca [Ringrose *et al.*, 1930–31] and Wisconsin [*loc. cit.*] the external symptoms appeared at about 3 weeks, after which, if the birds were continued on a severely "pellagric" diet, they died in a week or so. The former investigators, however, show a photograph of a bird which survived until 18 weeks of age, possibly on a less deficient diet. These authors used a ration composed of yellow maize 60, wheat middlings 20, bone meal 3, cod-liver oil 1, NaCl 0·5 and various supplements such as casein, egg albumin and autoclaved yeast so that when more than 15·5% of the supplements was added, the yellow maize was decreased by that amount. Each ration contained 25% of crude protein. The casein was either the commercial brand, or purified by being leached with acidified water. The egg albumin was Merck's product. In addition to the pellagra, a type of paralysis occurred which appeared to be related to the part of vitamin B

which is not destroyed by autoclaving. This paralysis affected the birds rather suddenly, causing them to lose partial control of their legs and to walk on their hock joints. The toes curled inward, never outward, and there was no slipped tendon. A summary of the results is given in the following table :—

Diet :	A.	B.	C.	D.	E.	F.	G.
Purified casein ...	15·0	10·5	13·0	14·25	15·0		
Autoclaved yeast ...		10·0	5·0	2·5			
Milk vitamin conc....	10·0						
Egg albumin ...						17·75	
Commercial casein ...							16·25
Approx. weight at 12 weeks (g.)	900	1,000	950	700	275	250	550
Degree of pellagra ...	—	—	—	+	+++ (25·9%)	++++ (100%)	++ (13·5%)
Degree of paralysis	+	—	+	++	++++	—	+++
Per cent mortality...	0	0	4	0	58·3	90·9	16·7

The most severe symptoms of pellagra were produced on diet F which contained egg white. With diet E, which contained the purified casein unsupplemented with autoclaved yeast or milk vitamin concentrate, growth was less rapid at first, and the symptoms appeared later. With commercial casein the symptoms were still less severe, and growth continued longer. 2·5% of autoclaved yeast in addition to the purified casein (diet D) was insufficient to prevent pellagra, but the condition did not occur with diets containing 5% or more of autoclaved yeast (diets C and B) or 10% of the milk vitamin concentrate (diet A). If it be assumed that the mode of preparation of the egg white destroyed the B_2 potency which Chick and Roscoe [1929] concluded it possessed, the development of the pellagra-like syndrome would seem to be related to a B_2 deficiency. That the " nutritional paralysis " was also a result of B_2 deficiency, however, is less clear, for while it was prevented by 10% of autoclaved yeast, it did not occur with the diet which contained egg white. Its non-appearance with the latter diet was apparently not related to the less rapid rate of growth, for the chickens grew faster on diet G than on diet E, and the degree of paralysis was less. That the anti-paralysis factor was not vitamin B_1 is suggested by the symptoms, and the fact that the condition was prevented by 10% of autoclaved yeast. A consideration of these facts and the circumstance that, before the appearance of pellagra the egg white was more " growth-promoting " than the purified casein, led the authors to conclude that autoclaved yeast contains at least two factors necessary for the nutrition of chickens.

The Wisconsin investigators also prevented the pellagra-like condition by means of autoclaved yeast. They considered chickens to be useful animals with which to test the activity of both B_1 and B_2 preparations: with such specific symptoms the error in an assay is less than when using a criterion like growth, which may be influenced by many factors. In their more quantitative work they found it necessary to add 6% of autoclaved yeast to the original diet No. 240 which had been dry heated at 100° C. for 144 hours. 8% of skimmilk powder or 0·8% of a liver extract also supplied sufficient of the antipellagric factor. They concluded that the chicken's requirement for B_2 is considerably higher than that for B_1. They did not observe the paralysis described by Ringrose, Norris and Heuser.

It is unlikely that the fundamental nutrition of the chicken will be discovered by the use of natural foodstuffs alone. This is borne out by the work of the Wisconsin investigators. They took the symptoms of paralysis and pellagra as criteria for deficiencies of the B_1 and B_2 factors respectively. They found that autoclaving at 120° C. destroyed B_1 and dry heating at 100° destroyed B_2. On their diets the chickens grew normally when these two fractions were added, so they concluded that chickens do not require any factor of the vitamin B complex which is destroyed at temperatures below 100° C. This excludes the heat-labile B_3 factor which Eddy, Gurin and Keresztesey [1930] concluded was necessary for chickens. These latter investigators found that chickens which were fed on a mixed grain ration which had been autoclaved, grew subnormally, and B_1 and B_2 concentrates did not improve the growth. Additions of the Williams-Watterman B_3 concentrate caused a gain in weight which was unaccounted for by increased food consumption.

However, when Keenan, Kline, Elvehjem, Hart and Halpin [1933] attempted to rear chickens on a "purified" diet of casein, dextrin, yeast, salt mixture and cod-liver oil they found that growth was retarded, and atrophy of the leg muscles coincident with paralysis occurred in about three weeks. The paralytic symptoms were the usual ones of "squatting" and muscular incoordination. The toes were either flexed or extended. Moderate amounts of natural foodstuffs did not reduce the incidence of paralysis below 20% but the addition of 18% of vacuum-dried, ether-extracted hog-liver resulted in normal growth and prevention of the paralysis. Two factors were considered to be present in the hog-liver: a thermo-stable growth promoting factor, and a thermo-labile (destroyed by autoclaving) anti-paralysis factor. The latter was thought to be very similar to vitamin B_4.

Summary.—Even in this short discussion of some of the work on the vitamin B requirement of chickens it can be seen that there are many problems awaiting solution. There is considerable similarity in the symptoms credited to deficiency of the various factors, but unless detailed examinations and reports are available the matter must remain obscure. "Paralysis" or "leg weakness," for example, seems to be one of the commonest symptoms

of ill health in chickens : as a general rule, however, there is some peculiarity distinguishing the different types.

The factors which are definitely known to be required are B_1 and B_2. Deficiency of the former causes the well-known polyneuritic symptoms. Under practical conditions with the common cereal mashes used, and with the birds allowed outside on grass runs, vitamin B_1 deficiency seldom occurs. Green food or the milling products made from the outer layers of grains will prevent the symptoms appearing in birds kept in confinement. Yeast or yeast preparations are commonly used in laboratory work. The case with regard to vitamin B_2 deficiency is not so simple of diagnosis. Seldom does one get symptoms of pellagra under even intensive commercial conditions ; it occurs only under fairly rigorous experimental control. Should the paralysis mentioned by Ringrose, *et al.* be considered to be a symptom of vitamin B_2 deficiency, it would seem that many cereal diets contain insufficient amounts of the vitamin. The cure is probably simple enough—lessening of the vegetable protein in the diet if it be already very high, the giving of milk and, in severe cases, of marmite or other potent yeast preparations.

While realising that work on the other factors awaits confirmation, we may tentatively consider that both B_3 and B_4 are required. The paralysis described by Ringrose, *et al* and by Kline, *et al* was rather similar but apparently due to different causes : in the former case it was prevented by the use of autoclaved yeast, and in the latter by a factor which was destroyed by autoclaving.

Vitamin C.

It has been shown quite often and fairly conclusively that chickens do not require vitamin C in their food [Mitchell, Kendal and Card, 1922–23 ; Plimmer and Rosedale, 1923 ; Carrick and Hauge, 1925 ; Hart, Steenbock, Lepkovsky and Halpin, 1925]. That they do require it for their metabolic activities, however, would seem probable, as the livers of chickens fed on a vitamin C-deficient diet may be quite rich in the vitamin. Bayon [1932] considered vitamin C deficiency might be a factor in predisposing chickens to disease. Holst and Halbrook [1933] described a condition which they considered to be like scurvy. The chickens were fed fish meal, yellow maize, yeast, sardine or cod-liver oil and oyster shell. The symptoms appeared from the third week onwards. They included nervousness, lameness and stiffness of the hocks, hæmorrhages from the pin feathers and skin, with hæmatomata under the skin. *Post mortem* examination showed hæmorrhages in the intestine and erosion of the gizzard lining, the latter occurring in 70% of the cases. The bones were brittle and the bone marrow dry and colourless. The hæmoglobin fell as low as 1 per cent. Fast growing birds developed the symptoms earliest. When skimmilk powder was fed in place of part of the yeast and fish meal, almost normal birds resulted. 5 g. of cabbage per bird was curative. The authors suggested that chickens are either unable

to synthesise vitamin C or do so in insufficient amounts. This is a preliminary report and it is difficult to decide whether the conclusions are entirely justified by the evidence. Fresh cabbage is certainly a rich source of vitamin C but dried skimmilk is not generally considered so. It will be remembered that hæmorrhages were frequent in the experiments of McFarlane, *et al.* (*vide* section on simplified diets) when fish meal, but not when buttermilk powder was included in the diet. The highest hæmoglobin was obtained when cod-liver meal was used. It may be, of course, that the fowl is able to synthesise ascorbic acid from other related acids, which in the experiments of Holst and Halbrook were present in inadequate amounts. The symptoms as described could fairly justifiably be ascribed to an acute anæmia, but conclusions cannot be made without further evidence.

Vitamin E.

This vitamin will be discussed in connection with hatchability. It will suffice to say here that from Card's experiments [1928–29] it would appear that under laboratory conditions better growth and lower mortality may result when chickens are supplied with this vitamin.

Minerals.

The mineral elements which have received most attention recently in the nutrition of the chick are Ca and P, and much research has been directed towards elucidating the optimum amount and ratio of the two in the ration. Earlier work had shown the effect of different mineral supplements on growth, and many investigators found apparently little difference in the availability of common mineral supplements for bone development, as judged by the ash percentage of the leg bones. There was some difference in the effect on growth in certain experiments, but Bethke, Kennard and Kick [1929–30] did not stress this, suggesting that it might have been due to some other factor. The data obtained, however, have not been entirely in harmony, owing possibly to different conditions at some centres compared with those at others, with regard to the type of basal ration, breed of chickens, etc. The results of Buckner and his associates [1928–29, 1929–30] seemed to show that $Ca_3(PO_4)_2$ was somewhat better than $CaCO_3$ or oyster shell, but at Wisconsin [Halpin and Lamb, 1932] this was not so with 2% or more of rock phosphate, due apparently to its relatively high content of fluorine [*vide infra*].

It would seem that logically the first step in the study of mineral requirements should have been an investigation of the optimal amounts of the various minerals in the ration, but stress seems to have been laid rather more on the optimal ratio of Ca : P. This can perhaps be understood because the

cereal rations commonly used are higher in P than in Ca, and as the ash of the leg bones has a ratio of approximately 2 : 1 it might seem of more importance to study the ratio of Ca and P in the ration. Again, in the study of rickets in rats, it was found that with a Ca : P ratio in the diet of approximately 1·6 : 1 rickets did not develop even with little or no vitamin D in the diet. Experiments were then planned to see if there were an optimum ratio of Ca : P for chickens, so that vitamin D would not have to be supplied. Thus in many of the trials at Wisconsin and Purdue the rations were unsupplemented with vitamin D, but under these conditions calcification did not proceed normally, regardless of the ratio or the quantity of Ca and P in the diets. The Wisconsin workers [Hart, Scott, Kline and Halpin, 1929–30] concluded that it was necessary to have a large amount of Ca in the ration. Thus the best growth of the chicks and the highest ash percentage of the tibiæ were obtained in one experiment with 3% Ca, a ratio of Ca : P = 8 : 1. With 0·35% Ca in the ration and a Ca : P ratio of 1·1 : 1 the bone ash percentage was very low and the rate of growth the poorest. Tully and his associates [1930–31] remarked that " Since vitamin D aids in the assimilation of calcium and phosphorus, correcting the deleterious effects of disproportions of these elements, it seemed advisable to conduct these studies [on the Ca and P requirements of growing chickens] chiefly in the absence of this influencing factor." The present discussion deals chiefly with results of experiments in which vitamin D in some form has been supplied, since it has not yet been possible to rear chickens to maturity with a total absence of the antirachitic factor. It is difficult to decide from the reports of experiments what was the exact nature of the pathological condition, and McGowan has frequently [*e.g.* 1933] insisted on the essential difference, ætiologically, pathogenetically and morphologically between the two conditions, rickets and osteoporosis. From the scientific aspect it is of interest to know if completely healthy chickens can be reared with a total absence of vitamin D but until this has been shown possible it is of greater importance from the point of view of the commercial poultryman to know the optimal amounts of Ca and P to supply in a ration when the vitamin is not a limiting factor.

In many cases comparative results are available where parallel experiments were conducted with and without the addition of vitamin D. The latter are inserted for interest, but it should be noted that in no case was growth optimum for more than a few weeks. Bethke, *et al.* [1928–29] concluded that a Ca : P ratio of 3–4 : 1 was optimum for calcification. It can be seen from their data that for the groups not receiving cod-liver oil the growth, on the whole, was better with ratios of 2–4 : 1 than with wider or narrower ratios. The best growth, nevertheless, occurred when the proportion was 1·75 : 1. When vitamin D was supplied, the best growth occurred with ratios of 1–2 : 1, the rate being less on either side of these figures, but falling away more rapidly with the wider ratios. If, however, their data are arranged in order of increasing percentages of Ca in the ration (the P percentages being somewhat similar in different groups), it can be

seen that there is a closer gradation in both growth and calcification, these being better for the no-cod-liver oil groups with increasing Ca percentages up to 3·5%. In groups receiving cod-liver oil growth was better with lower amounts of Ca. In these experiments the percentage of ash in the dry tibiæ, which was taken as the criterion for calcification, agreed fairly well with the growth. It would appear that the Ca : P ratio, *per se*, had less effect on growth or calcification than the level of Ca and P in the diet, the ratio occurring as a natural sequence.

Hart, *et al.* [1929–30] have also been cited as showing that wide ratios are of greater benefit in "growing" rations than narrower ones, the best ratio in the absence of vitamin D being about 8 : 1 (Ca : P). From their data, however, there would seem to be no close relationship between the ratio and either growth or calcification. Certainly in the first experiment reported in their paper, the best growth in the unirradiated groups was obtained with a Ca : P ratio of 8 : 1, but the two next best were with ratios of 2 : 1 and 1·75 : 1. In the irradiated groups there was a closer relationship, the optimum being with ratios of 1·5 : 1 to 3 : 1. When the groups are arranged in order of increasing Ca percentages in the diet, with the unirradiated groups the conclusion would seem to be that large amounts of Ca are required for growth, but not necessarily for calcification. With irradiation, again, the group receiving 0·71% Ca made the best growth, and the group receiving 0·9% Ca showed the highest ash percentage in the tibiæ, the gradation being on either side of these figures.

When vitamin D was supplied, and the P content of their rations was kept at about 0·44% Bethke and his associates [1929–30] found that with $CaCO_3$ additions the best growth and calcification up to 8 weeks occurred with 2% $CaCO_3$ in the ration, making a level of 0·86% Ca and 0·44% P, a ratio of almost 2 : 1. Wilgus [1930–31] came to somewhat the same conclusions; in his experiments the optimum growth occurred with approximately 1% Ca and 0·5% P in the ration. The minimum Ca required seemed to be about 0·9%, for while less than this resulted in good growth up to 6 weeks, at about 8 weeks there was some retardation of growth, with somewhat lower bone ash percentages. 0·5% P appeared to be quite sufficient if the ratio of Ca : P were not too wide. Under the conditions of the experiments it appeared that a Ca : P ratio of 1–2 : 1 was optimum, 2·5 : 1 on the border line, and 3·3 : 1 disastrous. In the experiments of Bethke *et al.* [1928–29] where Ca : P ratios of 3–4 : 1 were thought to be optimum, the P content was somewhat higher than in the experiments of Wilgus.

In discussing this question, Titus, McNally and Hilberg [1933] concluded that there was no common optimum ratio for every ration but that it depended in part on the metabolic acid-base equilibrium of the diet. Thus they found somewhat better growth resulted from the use of $CaSO_4$ than of $CaCO_3$, at the same level of Ca. From the growth curves given by Mussehl, Hill, Blish and Ackerson [1930] one might draw the conclusion that 2–9 week old

chickens can tolerate a potentially acid ration better than an alkaline one. Using a mash supplemented with 2% $Ca_3(PO_4)_2$ as a basis for comparison, they compared the effects on growth of additions of NH_4Cl or $NaHCO_3$, with and without $Ca_3(PO_4)_2$. Without the phosphate, 2% NH_4Cl resulted in slightly slower growth than did the basal diet alone, 4% in still slower growth, while 4% $NaHCO_3$ was more or less intermediate. The mortality with the latter addition, however, was exceedingly high. With 2% $Ca_3(PO_4)_2$ in the mash, addition of 2% NH_4Cl resulted in retarded growth, while the addition of 2% $NaHCO_3$ resulted, on the whole, in as good or slightly better average growth than without it. The mortality in one experiment was low, but exceedingly high again in another. This suggests that possibly the weaker chickens could not tolerate very well the potentially more alkaline ration and died, while those birds which could tolerate it grew well, thus bringing up the average body weight during the later weeks. With the NH_4Cl, however, the mortality was considerably less, and the resulting slower average growth might have been due partly to an actual effect when compared to the ration containing tricalcium phosphate, and partly to the survival of the slower growing birds when compared to the effect of the bicarbonate. Whether or not this conclusion is correct is difficult to say from these experiments, for it is reported in the same paper that while growth was about the same when 1% Ca was supplied as either $CaCO_3$ or $CaSO_4$, it was definitely worse when $CaCl_2$ was used.

A type of "leg weakness" in chickens often called "slipped tendon," "perosis" or "deforming leg weakness" is thought by some investigators to be due in part to an excess of minerals in the ration. Thus Herner and Robinson [1932] observed a greater incidence of leg abnormalities accompanied by "slipped tendons" in their experiments where the basal ration, containing meat meal and 2% of cod-liver oil, had meat meal ash added. The basal ration contained 4·73% of ash (1·4% of Ca and 0·85% of P), and when unsupplemented resulted in very few abnormalities. The addition of meat meal ash to raise the ash percentage of the ration to 6·31% (1·86% Ca, 1·10% P), resulted in slightly better growth, but over 70% of the birds had "leg weakness" at 12 weeks of age. The question of "leg weakness," however, will be discussed later.

When different sources of Ca and P are used in the ration, it is important to consider not only the levels of Ca and P and the effect on the metabolic acid-base equilibrium which will result, but also the effect of possible impurities. Thus while some investigators have found tricalcium phosphate to be somewhat superior for growth to other Casalts, Halpin and Lamb [1932] found that more than 1–2% of a certain sample of rock phosphate resulted in poor growth, and rather low percentages of ash in the tibiæ. This was roughly related to the fluorine content of the ration, as contained in the rock phosphate. Dolomite, also, contains a considerable amount of Mg, as also may certain limestones, and Mussehl, Hill, Blish and Ackerson [1930], Buckner Martin and Insko [1932] found that chickens developed a

type of " leg weakness " when the rations were high in $MgCO_3$, especially when $Ca_3(PO_4)_2$ was given as well. The effect was not so apparent when the Mg was given as the sulphate. This was not a rachitic condition, judging from the bone ash percentages and the shape of the bones.

It would seem that in areas known to be iodine deficient and when iodine-containing foods are not used, this element should be supplied in some form. Hamilton and Kick [1930] found that under their conditions no benefit was derived from the addition of KI to the ration. The iodine content of the food was not reported, however, so their results do not argue against the use of iodine in certain areas for growing birds. That poultry can withstand considerable amounts of iodine was shown by Orr, Crichton and Middleton [1929] and others [Schmidt, 1932, 2 ; Sharrer and Schropp, 1932], although Brown [1931–32] stated that hens refused to eat mash containing about 0·2% KI.

It is generally accepted that NaCl should be supplied in the ration, the common practice being to put 0·5–1% in the mash. Indeed, from the experiments at the Rowett Institute and at Belfast it might appear that when birds have access to grass runs, and oyster shell *ad lib.* NaCl is the only other mineral supplement necessary, provided the extracted soy bean meal content of the ration is not too high. The toxicity of salt is apparently only evident when the birds consume large quantities [Hamilton, Card and Carman, 1926 ; Mayall, 1929]. This they will not ordinarily do, but in one instance mentioned [Mayall] the salt was accidentally included by mixing salted beans with the wet mash which was given to birds which were quite hungry. Symptoms were listlessness, excessive thirst, inflammation of the digestive tract and yellowish droppings. The mortality was high, especially so, probably, because the water trough became empty.

In Scotland and N. Ireland extensive studies have been made of the mineral requirements of chickens. Discussion of these experiments has been deferred because their purpose has been not so much to discover the fundamental mineral requirement, but rather the best kinds and amounts of supplements to use under poultry-farm conditions where the birds have access to oyster shell and grass runs, at least after the first few weeks subsequent to hatching. It can easily be seen that while under indoor conditions a certain supplement may give far better results than another, this need not necessarily be the case when birds have access to good grass runs, and *vice versa*. The experiments have been reported in several papers. Briefly, it was found that when a " complete " mineral mixture—such as had been advised for pig rations in the earlier work, or one comparable to the mineral composition of milk in more recent work—was used, the birds grew considerably better than without the mineral supplement. The " growth-promoting " effect was almost equivalent, in a cereal ration with extracted soy bean meal, to that of liquid skimmilk. This effect had also been observed by Kennard, Holder and White [1921–22] in America.

The results warrant discussion in greater detail. In one series of experiments [Baskett, 1931 ; Prentice and Baskett, 1931] the basal ration was composed of bran 2, maize meal 1, Sussex ground oats 1 and sharps 1 part. Each group was supplied with oyster shell *ad lib.*, and had access to grass runs. Where a mineral mixture was given, it consisted of steamed bone flour 72, NaCl 11, KCl 14·25, sulphur 2, Fe_2O_3 0·75 and KI 0·07. The chickens receiving the basal ration did poorly, grew slowly and matured late, with a low body weight. The pullets laid well, however, once they commenced. By supplementing the basal ration with the mineral mixture, growth was considerably improved, mortality lowered and the pullets matured far earlier although with no greater body weight than without the mineral addition. Egg production was good. The addition of fish meal or meat meal to the basal ration resulted in more rapid growth, with age at maturity approximately the same as the group receiving basal + minerals. Body weight at maturity, however, was considerably greater, although subsequent egg production was not. In the groups receiving liquid skimmilk *ad lib.*, or 18% soy bean meal + 4% minerals in addition to the basal ration, there was the most rapid growth, and the pullets were the heaviest at maturity : age at maturity was little different from the group receiving the basal + minerals. Egg production was good, but the question of the effect of the diet on the weight of eggs was somewhat complicated by the fact that in one experiment the heaviest eggs were laid by the group receiving the basal ration alone.

Under the conditions of the experiments, then, the best results were obtained by supplementing the basal ration with either liquid skimmilk *ad lib.*, or 18% soy bean meal + 4% minerals, the latter supplement probably being the cheaper for most poultrymen. The conclusion reached was that whereas the amount of protein in the ration might affect the weight of the birds at maturity, " it is the mineral, and not, as is frequently supposed, the protein content of the ration which determines the rate of maturity " [Prentice, Baskett and Robertson, 1930]. It was realised, of course, that the experiments were designed for the purpose of compiling rations suitable for the commercial poultry farmer and not primarily to elucidate the fundamental nutrition of the fowl. Thus it was specifically stated in reference to the greater food consumption per unit live weight gain of the group receiving liquid milk compared to the other groups (basal alone and basal + minerals) that the " explanation may be that grass and insects represented a greater part of the total food consumed by the other groups, since they were far more active foragers. . . . The extent to which grass may affect the protein requirements is another point worthy of general investigation " [Prentice, 1933, 1]. The results show the benefit which the poultryman in Scotland and N. Ireland may obtain from the judicious use of minerals in his poultry rations. A further and full account of the work is given by Prentice [1933, 2]. The data indicate that under conditions similar to those in the previous experiments where birds have access to good grass runs and

oyster shell *ad lib.*, the mineral mixture may be considerably simplified without deleterious effects. " As far as the rearing and growth of the chicken and the mortality rate are concerned no ill effects followed the removal of the steamed bone flour . . . the sulphur and the iron and the iodine, but the removal of the two chlorides was disastrous in its result " [Robertson, 1930]. From observations on the rearing of chickens at both centres (Scotland and N. Ireland) it was indicated that Na was the important element in NaCl for $NaHCO_3$ gave results similar to NaCl while glycine hydrochloride promoted no better growth than the unsupplemented basal ration. " The presence of $\frac{1}{2}$ per cent. common salt in the mash supplied to young chickens is essential for satisfactory growth, resistance to disease, economical utilisation of feed and early maturity in the pullets. No other mineral supplement is necessary, apart from lime. . . . When $\frac{1}{2}$ per cent. of common salt is used as a supplement to the mash of laying hens as good results in terms of egg production, value of eggs laid, size of egg and health of the birds are obtained as from the use of a complex mineral mixture of steamed bone flour, common salt, muriate of potash, iron oxide, sulphur and potassium iodide " [Prentice, 1933, 2]. The results of these experiments are naturally only applicable where similar conditions exist. Where birds are kept indoors, as in Canada during the winter, for example, it is unlikely that the same results will be obtained.

" Leg Weakness."

Some of the reports dealing with the condition of " leg weakness " of poultry are reviewed in this section. The term " leg weakness " is employed because it is sufficiently vague not to indicate any specific condition of the legs. It would appear that much of the confusion in the literature has been a result of too little attention being paid to other than gross macroscopical manifestations. As was pointed out by Stafseth and Mallman [1928–29] and others [Thomas, 1931 ; Henderson, 1933], " leg weakness " or even paralysis are not specific symptoms but may be caused by a number of different agents. The following are some of the more or less distinct forms of " leg weakness," classified as such by means of causal agent, curative agent or characteristic symptoms.

A.—Due to infestation by intestinal parasites.

B.—Due to infection.

1. Non-specific symptoms or lesions (as regards the limbs), observed in cholera, leucæmia, *etc.*
2. More specific symptoms and lesions, observed in " fowl paralysis " (*neurolymphomatosis*).

C.—Due to dietary errors.

1. Deficiency of vitamin D—" rickets," etc.
2. Deficiency of calcium.
3. " Slipped tendon," " perosis."
4. Excess of magnesium.
5. " Nutritional paralysis " of Norris, Heuser and Wilgus [1929–30].
6. Deficiency of vitamin B_1—polyneuritis.
7. Deficiency of vitamin A.

In a consideration of the symptoms, cause and cure of a particular type of " leg weakness," it is clearly necessary to bear in mind that there are often different contributory factors which may complicate the diagnosis. Thus it would seem that the *neurolymphomatosis* type of paralysis occurs so often in conjunction with worm infestations or with coccidiosis that the infestation is considered by many writers to be the cause of the typical nerve lesions. Some types of " leg weakness," on the other hand, appear to have symptoms sufficiently characteristic for the diagnosis to be relatively simple. In the review of the types of leg abnormalities which appear to be a result of dietary errors the attitude is taken up that malformation of the bones is not in itself sufficient evidence of a rachitic condition. If one acceded to the suggestion of Henderson [1933] that all deformities of the bones should be called rickets, considerable confusion would result in a discussion of the subject.

A.—" Leg Weakness " Due to Infestation by Parasites, etc.

Infestation by different species of worms and by protozoa very often results in emaciation and weakness. Some of the poisons produced are said to cause " lameness, droopy wings, lack of co-ordination of movements, paralysis, wry neck and blindness " [Graham and McCulloch, 1928]. This paralysis is seldom to be confused with that caused by dietary errors. The diagnosis of coccidiosis and of worm infestations is fairly simple as a rule—by the bloody droppings, the condition of the caeca in coccidiosis and, if necessary, a microscopical examination of the intestinal contents. Bayon [1932], however, considered that tape worms may cause paralysis which is often confused with the *neurolymphomatosis* type. The prevention and cure of infestations are fully discussed in papers and books dealing with the subject, and need not be considered here.

B.—" Leg Weakness " Due to Infection.

1. *Non-specific symptoms or lesions.*

This group includes those diseases like cholera and leucæmia, *etc.*, which result in ill health and weakness of such a degree that the affected birds

become too weak to stand or walk, and may occasionally lie on one side. Dullness and listlessness are common, and as a rule the appetite is impaired. The "leg weakness" is seldom to be mistaken for one caused by dietary deficiencies but is not dissimilar to some cases associated with food poisoning, or with so-called "true fowl paralysis" [Bayon, 1932]. Correct diagnosis is facilitated by other manifestations of the disease.

2. *More specific symptoms and lesions—"fowl paralysis."*

The literature [*vide* Biely and Palmer, 1932, for a review] on fowl paralysis, range paralysis or *neurolymphomatosis gallinarum* is voluminous, but there is general agreement by writers as to the characteristic symptoms. The onset of the paralysis may be relatively rapid, and may occur between the third and eighteenth months after hatching, although it has been observed as early as the 37th day [Biely, Palmer and Lerner, 1933]. Typically, there is drooping of the wings, lameness and subsequent paralysis of one or both legs, iritis and often blindness, and not infrequently twisting of the neck. The appetite usually remains keen and the birds may live quite a long time if they are fed by hand. The later stages of the disease result in complete prostration. On *post mortem* examination, lesions are found of the peripheral nerves and nerve ganglia, especially in those of the limbs. Sarcomatous tumours are common in the internal organs, and accumulations of lymphocyte, mononuclear or plasma cells in the nerve ganglia are the rule. In his discussion of the macroscopic diagnosis of fowl paralysis Jungher [1933] considered that thickening of the dorsal root ganglia of the brachial plexus was more commonly found than lesions of the sciatic plexus.

The ætiology of uncomplicated *neurolymphomatosis* is uncertain. It has been ascribed to a virus, the evidence being that a certain percentage of transmission experiments have been successful, and that it appears to be transferred through the egg, so that certain strains show a greater incidence than others. The disease is not highly infective, however, as healthy birds kept in close confinement with affected ones may not contract the disease. That it is often accompanied by other diseases is evident, and care is required in diagnosis.

Bayon [1932] considered it very likely that this type of paralysis could be cured by forcibly feeding affected birds with lettuce. In his review he discussed the differential diagnosis of various forms of "fowl paralysis" caused by infestation or infection and concluded that while the virus theory was probably correct in part, there seemed to be strong evidence suggesting that there were many contributory factors. He advised the feeding of green-stuffs to farm flocks.

The disease occurs in many parts of the Empire: it has been observed in N. Nigeria [Hall, 1932], Cyprus [Manley, 1932] and Canada [Biely, *et al.*, 1933] as well as in Great Britain [Warrick and Dalling, 1932] and N. Ireland [Kerr, 1931-32]. Lettuce feeding has not been uniformly successful. Blount

[1932] held little brief for the virus theory but was of the opinion that important contributory factors were a deficiency of flint grit or of fibre, or an excess of vitamin B. He submitted scanty evidence, however, and it is doubtful, in view of investigations on the role of grit, fibre and vitamin B if confirmatory proof could be obtained to show these factors are connected with the origin of this type of paralysis. It is not improbable, however, that the nutrition of the fowl may influence its resistance to certain types of infection.

C.—"Leg Weakness" Due to Dietary Errors.

1. *Deficiency of vitamin D—"rickets," etc.*

The symptoms, prevention and cure of vitamin D deficiency have been discussed in the section on Vitamins (*q.v.*) but a few points will be repeated here for clarity in differentiating the various forms of "leg weakness." That under present conditions some form of vitamin D is necessary for the successful rearing of chickens has already been mentioned. The same is true of adult fowls, but laying pullets succumb more rapidly than non-layers or cocks. It is preferable to call the condition "vitamin D deficiency" so as to include not only rickets but other diseases which are alleviated or cured by vitamin D therapy.

The hock joints may enlarge but distortion of the toes or the "slipped tendon" appearance is infrequent in uncomplicated rickets or other forms of vitamin D deficiency. Post mortem examination may show beading or "waving" of the ribs, crooked breast bones and softness of the limb bones. The parathyroids may be enlarged. Laying pullets show a somewhat similar ruffling of the feathers and distortion of the breast bones and in very advanced cases pliable or brittle leg bones. The birds often become so weak that they walk with difficulty or not at all, and may die in a very emaciated condition. Bending of the leg bones is a much cited symptom, but it would appear that not infrequently an incorrect diagnosis of rickets has been made on this criterion. The more general term of "vitamin D deficiency," however, undoubtedly does include cases which show bow-legs. In mature females the production of soft-shelled eggs is often, but not always, an indication of vitamin deficiency. The chief symptoms are the fragile nature of the bones and beak, beading of the ribs and enlargement of the parathyroids.

Preventive measures for chickens or adult birds which are kept indoors include the use of at least 0·5% of cod-liver oil in the food (1% in the mash if grain be given also). Slightly greater percentages of cod-liver oil are indicated in the initial stages of the cure.

A great excess of vitamin D will also cause "leg weakness," but as it is unlikely to be met with under commercial conditions, it will not be discussed here.

2. *Deficiency of Calcium.*

In young chickens the adequacy of a given amount of calcium is dependent to a considerable extent on the presence or absence of vitamin D. When the vitamin is not supplied, increasing quantities of calcium (within limits) may result in improved rate of growth although not necessarily of bone ash percentages (*vide* section on Minerals). The converse, however, is not true. The mode of prevention is self-evident. For most types of " growing " rations the addition of calcium-supplements to make a total of about 1% Ca in the mash is probably adequate when vitamin D is supplied. Salts like $CaCO_3$ would seem preferable to $Ca_3(PO_4)_2$. It would appear to be unnecessary to increase the calcium content of the ration for the sole purpose of " balancing " phosphorus which, while harmless, may yet be present in very considerable quantities in such foodstuffs as soy bean meal [*cf.* McGowan, 1933, 1, 2]. It is often advantageous to allow free access to supplements like oyster shell. For laying pullets and hens the view of many investigators is that a plentiful supply of oyster shell is all that is required as a source of calcium when vitamin D is not a limiting factor.

3. *" Slipped tendon," " perosis."*

This type of paralysis has a number of synonyms. In Canada it is called " slipped tendon " and not infrequently " range paralysis " : in the United States it is also called " hock disease " and " perosis," and in Great Britain some investigators speak of it as " deforming leg weakness." The symptoms have been noted in chickens but not in mature birds, and formerly were confused with those of rickets. There is some ruffling of the feathers and bending of the legs, etc., but almost invariably the tendon at the back of the " hock " joint slips to one side or the other and the shank bone of the leg (the *tarso-metatarsus*) is twisted to one side, generally outwards, and may rotate backwards. Frequently only one leg (often the right) is affected, so that the chickens can hop about quite actively on the healthy one. When both legs are deformed, the birds walk on their " hock " joints. Males seem more susceptible than females, and the heavier breeds more than the lighter ones. Except in the terminal stages of the disease the appetite is usually unimpaired, and the birds are quite active. It is exceptional for the toes to turn inwards unless there are other complications. Chickens may be affected from three weeks onwards almost to maturity, and growing birds on range have been observed with a very similar, if not identical condition. The symptoms are sufficiently different to prevent confusion with rickets. The blood serum-Ca and inorganic-P, bone ash and bone phosphatase values are normal [Hall and King, 1930–31] and neither cod-liver oil nor irradiation are of value as preventive or curative measures. Titus [1932] found that the epiphyses and the diaphyses of the tibiæ remained disunited at 12 weeks of age, although the epiphyses appeared more than normally calcified.

Hunter, Dutcher and Knandel [1930–31] were able, by feeding rations high in bone meal, sodium phosphate, calcium carbonate or so-called balanced mineral mixtures, to cause 90% of the chickens to develop " slipped tendon." Decreasing amounts of mineral supplements resulted in decreasing incidence of abnormalities. Oats or oat feed, however, possessed beneficial properties.

With a ration containing 5·7% ash, 1·42% Ca and a Ca : P ratio of 1·54 : 1, Titus [1932] obtained 42–85% abnormal chickens. When the 3% bone meal of this ration was replaced by $CaCO_3$, the Ca : P ratio being widened to 3·24 : 1, 10% fewer cases resulted in one experiment. The addition of 10% of rice bran to these rations lowered the Ca : P ratio to 1·34 : 1 and 2·51 : 1 respectively, and decreased the incidence of " perosis " to 12% and 0% respectively. Replacing the bone meal by either $CaHPO_4$ + $CaCO_3$ or NaH_2PO_4 + $CaCO_3$ to supply equivalent amounts of Ca and P, did not modify the development of the " perosis." 10% of rice bran in a suitable ration prevented the occurrence of the condition but in the absence of cod-liver oil did not prevent " rickets." Rice bran is rich in P, but according to Titus it was not the P, *per se*, which was beneficial, nor the zinc present in the bran. An ether extract was of no benefit, but it appeared to the author that possibly a factor in the vitamin B complex was concerned. Titus considered that " when adequate quantities of the necessary food factors are present and the dietary acid-base balance and the calcium-phosphorus ratio are approximately optimum, a mere excess of mineral ingredients does not cause a higher percentage of cases of ' enlarged hocks ' or ' slipped tendon,' *i.e.*, perosis, than is observed when much less mineral supplement is included in the diet " [Titus, 1932].

Experiments which are designed to show whether the mineral content of the ration or the Ca : P ratio are the more important factors in causing " slipped tendon " require to be carefully planned, for in changing the Ca : P ratio the ash content of the ration is often changed. From their experiments Payne, *et al.* [1932] concluded that excessive amounts in the ration of either Ca or P or both were important ætiological factors. The results of their fourth experiment do not indicate a close connection between the ratio of Ca : P or the amount of Ca, *per se*, in the diet and the incidence of malformed legs. The relationship between the per cent. of ash in the diet and the per cent. of abnormalities is close, the higher ash percentages corresponding to the higher percentage of " slipped tendons." The relationship with the percentage of phosphorus in the diet, however, is a closer one.

Herner and Robinson [1932] varied the amount of meat meal ash in rations for chickens so that the total ash percentage was from 4·7% to 7·1%. The Ca content was from 1·4% to 2·1% and the Ca : P ratio approximately the same in the four groups. The following table gives a summary of their results.

RELATION OF " SLIPPED TENDON " TO ASH CONTENT OF RATION.

[Data of Herner and Robinson, 1932.]

Number of group :	1	2	3	4
% ash in ration	4·73	5·52	6·31	7·10
% Ca in ration	1·40	1·64	1·86	2·10
% P in ration	0·86	0·98	1·10	1·22
Ratio Ca : P	1·63	1·67	1·69	1·72
Abnormal birds, 6 weeks	0	6	13	12
Abnormal birds, 12 weeks	4	23	31	17
% abnormal birds, 12 weeks	9·5	58·7	72·1	70·9

There was little difference in the analyses of the bones of normal and abnormal birds. The authors state that the condition had been observed more often in the birds receiving meat meal as a protein supplement and not at all when casein was used. The crippled condition was characterised by " digestive disturbances, followed by thick hocks, bow legs, short shanks, twisted legs, and slipped tendon. These abnormalities were permanent." As the Ca : P ratios of the four rations were very similar, the conclusion was that the amount of minerals was the important factor in the ætiology of this type of " leg weakness."

In a later paper Titus *et al.* [1933, 2] concluded that there was no optimum ratio of Ca : P for all diets, but that the metabolic acid-base balance of the diet was one factor involved in the development of " leg weakness " (which did not refer to the " slipped tendon " condition alone). Rations containing $CaCO_3$ or $CaSO_4$ were compared and it was found that whereas the latter salt resulted in a higher ash percentage of the leg bones, there was little decrease in the incidence of " perosis."

Parkhurst and McMurray [1933] concluded that the use of an excessive amount (over 9%) of meat and bone meal (35–40% ash content) in the ration for chickens was an important factor in the development of " perosis " or " deforming leg weakness." With little or no meat meal few cases occurred under their conditions. Ground limestone was not an important factor. From some of their experiments they deduced that " the proportion of calcium to phosphorus might be of greater importance than the amount of these minerals present in the ration," but it is not clear that the wider ratios were always less faulty than the narrower ones. " Slipped tendons " occurred, however, with rations containing only 3·77% total ash, and the condition was no more prevalent when ground limestone was added to bring the ash percentage up to 7·6%, the Ca : P ratios being 0·39 : 1 and 4 : 1 respectively. It is somewhat remarkable that in all their experiments there were so few

chickens that could be classed as normal. Neither the protein content of the ration nor the rate of growth materially affected the incidence of " perosis." That battery brooders appeared to intensify the bone abnormalities might be related as much to the lessened possibilty of coprophagy as to the mechanical effect of the wire floors. In some of their experiments they observed a condition of " crooked toes," but it is difficult to decide from their description if this were similar to that described by Norris, *et al.* [1929–30 ; 1930–31] : it was not prevented by 21% of dried skimmilk in the ration.

From the above short review of the subject it can be seen that the evidence is somewhat conflicting in regard to the ætiology of " slipped tendons " or " perosis." That it is related in part to the mineral content of the ration seems very probable ; it may occur, however, when this is quite low. The effect of the Ca : P ratio of the ration, *per se*, is less clear, although it may be that there is an optimum ratio for a given ration when other factors are also considered. The beneficial effects of oats and of rice bran are interesting, and point to something more than a mere mineral imbalance. For practical purposes it would seem inadvisable to use an excessive amount of mineral in the ration. In this connection it was pointed out by Graham [1932] that when chickens were given a free choice of different foodstuffs " none of the chicks . . . had any trouble with crooked legs or slipped tendons. It is rather remarkable that out of a number of over twelve hundred birds there was not a single bird with a trouble of this kind . . . The general results in the consumption of minerals, such as bone meal, oyster shell and granite grit, would lead one to believe that many of us have used too much mineral, and that it might be wiser to place such feeds in hoppers rather than to mix them in a mash." The average consumption of minerals by 5 groups of chickens, expressed as per cent. of the total food intake for the first six weeks, was as follows :—

	Group 1.	Group 2.	Group 3.	Group 4.	Group 5.
Oyster shell	0·91	0·58	0·47	0·30	0·22
Granite grit	1·00	0·13	0·47	0·35	0·12
Bone meal	0·61	0·20	0·14	0·24	0·01

Branion [1933 ; also personal communication] is of the opinion that too little attention is paid to the " cereal effect." Chicks have been reared on apparently adequate diets but with one or other of the common grains as the sole cereal. One such diet was made up as follows : cereal 100, buttermilk powder 10, fish meal 7, alfalfa leaf meal 5, bone meal 1, oyster shell 2, iodized salt 0·5 lbs. and cod-liver oil 1 pint. The general findings are that with yellow maize as the only cereal there is a high incidence of leg deformities, bent or bowed legs and slipped tendons. Barley produces

a porous, spongy bone but with few crooked legs. Wheat germ, whole oats, oat hulls and germ are largely preventive. " This condition has apparently no association with the antirachitic vitamin as now understood, nor directly with the calcium-phosphorus ratio, nor with the total mineral content of the rations, although the latter, if excessive, apparently aggravates the condition."

The solution of problems related to the calcium and phosphorus content of rations is not without difficulties. Because the incidence of " slipped tendons " is markedly different with two rations which contain similar amounts of phosphorus (*vide supra* the experiments of Titus), this is not in itself sufficient proof that the fault does not lie in the phosphorus content of one of the rations. Mention of the change in viewpoint from the importance of total dietary protein to that of quality of protein is in almost every text book on animal nutrition. The addition to a basal ration of sources of phosphorus, such as bone meal ($Ca_3(PO_4)_2$), $CaHPO_4$ and NaH_2PO_4, could very conceivably have quite different results from the addition of rice bran, in which the phosphorus is largely if not wholly in organic combination. This point has been stressed by McGowan [1933] in his discussions on calcium and phosphorus metabolism, and might well receive the attention of poultry investigators as a possible explanation for the apparent divergence in their experimental results.

There are many investigators who consider that the explanation of this abnormality lies in a deficiency of an unrecognised vitamin. While wheat germ middlings, untreated, was found at Guelph [Graham, *et al.*, 1934] to be preventive, acid extraction of the middlings resulted in a large number of cases of abnormalities. Brewers' yeast was not protective [Branion, 1933]. One cannot as yet be really dogmatic in stating whether this abnormality can be explained on a vitamin or on a mineral basis. It would appear that with the extensive experiments being carried out at Guelph and other centres this problem may be on the highroad to early solution.

4. *Excess of magnesium.*

Buckner, Martin and Insko [1932] have described a type of " leg weakness" occurring in chickens receiving large amounts of magnesium in otherwise adequate rations. The symptoms appeared at the end of the third week, when the birds " became nervous and weak, and the joints of their legs swollen. The swelling of the joints increased until the end of the sixth week when all the chicks of this lot were unable to stand erect and only moved when disturbed. Their toes turned out and they walked on the distal end of the tarso-metatarsus." The ration given to the group referred to contained 1·73% Ca, 1·40% Mg and 1·15% P. With less P but similar amounts of Mg in the ration the condition was less severe, improved spontaneously, and almost disappeared in one group by the eighth week. When the $MgCO_3$ was omitted from the ration of the group receiving 1·40% Mg

and 1·15% P the leg condition improved considerably. The " leg weakness " was believed to be different from " slipped tendons " because " the condyles at the distal ends of each tibia were in proper alignment with the shaft, which was straight." The shank bones were somewhat flattened and twisted, and the head of the tarso-metatarsus was at a decided angle to the shaft. At the end of six weeks the ash percentage of the leg bones was lower in the $MgCO_3$ and $Ca_3(PO_4)_2$ group than in the other groups, and at eight weeks the ash percentage was still low in those birds which had received magnesium. The practical value of these experiments lies in the suggestion that it is inadvisable to use limestones which contain high percentages of magnesium, such as the dolomitic limestones. Especially might this be so if rock phosphate were also given.

5. *" Nutritional paralysis."*

The symptoms of another type of " leg weakness," the " nutritional paralysis " of Norris, Heuser and Wilgus have been described by the Cornell [Norris, *et al.*, 1929–30 ; 1930–31] and Ohio [Bethke, *et al.*, 1930–31] investigators as occurring in chickens after about the third week from hatching. The birds lose partially the use of their legs rather suddenly and frequently walk on their hocks with the toes curled inwards and compressed together. They may walk on the distal extremities of the metatarsi. In advanced cases the chickens often lie on their breasts with their legs sprawled in opposite directions. The leg muscles are flabby and the skin somewhat dry, but without lesions. There is neither enlargement nor flattening of the hock joints, bending of the legs nor displacement of the tendons, as in " perosis." The curling inwards of the toes is an important symptom in differential diagnosis. In the earlier stages, the birds may be quite active, though awkward. Spontaneous partial or complete recovery is evidently quite common. This type of " leg weakness " may occur with rations adequate in regard to calcium, phosphorus and vitamin D. Blood serum-Ca, inorganic-P and bone ash values are normal. It frequently occurred when commercial or purified casein, menhaden fish meal, meat meal or dried whale meal were used as the sole source of animal protein : different samples of the last two products, however, varied in their effect. In general, the condition was prevented by adding to the ration sufficient quantities of a milk vitamin concentrate, dried dairy products (skimmilk, buttermilk or whey), brewers' yeast, autoclaved yeast, alfalfa leaf meal or dried egg albumin.

From a consideration of these foodstuffs, it might appear that a deficiency of a factor in the vitamin B complex which is not destroyed by autoclaving was responsible for the " leg weakness." In the discussion of vitamin B it was mentioned that Ringrose, Norris and Heuser [1930–31] observed the same type of " nutritional paralysis " with diets containing commercial or purified casein. Under the conditions of their experiments 10% of a milk vitamin concentrate or 5% of autoclaved yeast were insufficient to be entirely

protective. With diets containing 10% of autoclaved yeast or 17·75% of dried egg albumin, however, paralysis did not occur, although pellagra-like symptoms appeared with the egg albumin supplement. The Wisconsin investigators (*vide* section on Vitamin B) did not obtain paralysis in their experiments on pellagra-producing diets for chickens. They were able to destroy the pellagra-preventive factor by dry heat at 100° C. for 144 hours. It is possible that the pellagra-like condition sufficiently retarded growth to mask a possible deficiency of the supposed paralysis-preventive factor, and without further proof it would not be justifiable to state that the latter factor was not destroyed by the dry heat treatment. The variability of different samples of menhaden fish meal and meat meal is suggestive in this connection. The Wisconsin workers considered that the type of paralysis they credited to lack of vitamin B_4 might have been the same as this "nutritional paralysis." They found, however, that autoclaving destroyed the B_4 or anti-paralysis factor whereas in their earlier work Ringrose, *et al.*, had shown that it was present in their autoclaved yeast.

It is difficult to be certain whether the "crooked toes" condition observed by Parkhurst and McMurray [1933] was the same as this type of "nutritional paralysis." Very similar conditions have been observed, however, in Scotland and N. Ireland and possibly in England [Johnston, Kinross, Macdonald and Prentice, 1933]. These outbreaks have generally occurred with rations containing extracted soy bean meal as the chief protein supplement, and sometimes appeared even when the birds had access to grass runs. When she used a basal ration containing 18% of extracted soy bean meal, Kinross found that additions of 10% dried milk or 10% dried yeast did not prevent the "leg weakness." With a similar basal ration but omitting part of the soy bean meal so as to keep the total crude protein of the basal, the 10% dried milk and the 10% dried yeast rations, respectively, the same, it was found at Belfast and at the Rowett Institute that "leg weakness" occurred only in the basal group to any marked degree. Marmite and liquid skimmilk were found at the latter Institute to cure even fairly advanced cases. When 7% or less of soy bean meal was used, or when the protein supplement was a mixture of dried skimmilk, fish meal and soy bean meal, "leg weakness" seldom occurred. It has been suggested, also, that samples of soy bean meal may vary in their effect, since different varieties and methods of extraction are used in the preparation of the meal [Prentice].

Bethke and collaborators [1930-31] found fresh greens effective in preventing this nutritional type of "leg weakness." That the same or a very similar condition has been observed in birds having access to grass runs may be related to the fact that chickens which receive a high percentage of vegetable protein in their rations may eat very little grass, while those receiving the same percentage of protein but largely from animal sources may eat their runs almost bare of grass [Johnston].

From a practical standpoint it would seem inadvisable at present to include in the growing chick ration large quantities of foodstuffs believed to

be poor in the supposed paralysis-preventive factor, unless sufficient quantities of protective foods are added.

6. *Deficiency of vitamin B_1—polyneuritis.*

Under commercial conditions the polyneuritic type of "leg weakness" is met with relatively seldom, although it has been studied in laboratories. It has been discussed in connection with vitamin B_1 and will not be considered in great detail here. Symptoms include muscular incoordination and loss of balance and birds may be found sprawled in a corner of the pen or in a sitting position with their legs stuck out in front of them. In advanced cases there may be some resemblance between polyneuritis and the "nutritional" paralysis, but in the former condition the toes do not turn inwards and the birds seldom walk on their shanks. Adult birds are as often affected as chickens, and the typical opisthotonic position seen in pigeons used in vitamin B_1 assay may occur [Carrick and Carr, 1925–26].

Rations in general use contain considerable amounts of vitamin B_1 from bran, ground whole grains and green stuffs, etc. Yeast and yeast preparations have been used as curative measures.

7. *Vitamin A deficiency.*

Deficiency of vitamin A may also cause staggering and muscular incoordination and result in the birds lying sprawled on one side, but as a rule such marked symptoms seldom occur in chickens kept under ordinary commercial conditions. It is unnecessary to repeat here what has been said in the section on vitamin A.

Summary.—In differentiating the numerous forms of "leg weakness" in chickens and adult fowls it is not always enough to consider the symptoms alone, as the early stages of many of the types are rather similar. A knowledge of the previous history of the birds as regards food and environment is of great value. Also, there may be a complication of two forms of "leg weakness" in the same flock, for instance the "slipped tendon" and the "nutritional paralysis" types, as observed by Bethke, *et al.* [1930–31]. As with other diseases, early diagnosis may be relatively simple: but where the ætiology of the disease has not been fully investigated, one may have to try different curative measures before arriving at the correct one.

There are at least seven types of "leg weakness" which are definitely due to dietary errors. Their prevalence varies in different localities. Those caused by relative deficiencies of the vitamins (A, B_1 and D) and of calcium, while met with fairly often under other than strict laboratory conditions, are quite easily cured and prevented. Excess of magnesium in the ration is not a very common occurrence in most places. The "slipped tendon" and "nutritional paralysis" types of "leg weakness" may occur rather unexpectedly and are perhaps less easy to rectify. For the former condition, decreasing the amount of bone meal in the ration and adding rice bran (if

easily available) or oats or wheat germ middlings are remedies suggested by investigators. Dried yeast is becoming quite a common ingredient in many poultry food mixtures, and should assist in preventing "nutritional paralysis." A better method may be to give the birds liquid skimmilk to drink, or to increase the amount of dried milk while decreasing some of the other protein concentrates in the ration.

As was pointed out at the beginning of this section, "leg weakness" in poultry is by no means a specific symptom. It is not suggested that the forms reviewed here are the only ones which may be encountered under experimental or even commercial conditions.

Mineral "Balance" Experiments.

Retention of calcium and phosphorus by fowls has been investigated but slightly. As in mammals the study is complicated by excretion of the minerals into the intestine, and in fowls there is the further complication that the urine and fæces are voided together. Again, in calcium balance experiments with fowls, care has to be taken to ensure there is no reserve of calcium in the gizzard in the form of oyster shell or grit. The presence of so-called insoluble grit probably does not appreciably affect the results, but there is the possibility of small particles of grit being excreted and interfering at times with the accuracy of some of the determinations. The ideal method possibly would be to rear birds in wire cages so that there would be no chance of them ingesting any grit-like material. However, Common [1932] found that a pre-experimental period of 8 days was sufficient for pullets which had been taken from free range conditions to rid themselves of this source of fallacy.

Another consideration in balance experiments is the care which must be exercised in feeding the birds so that none of the food will be dropped on to the collection trays. The plan adopted by Common [1933] is to make the mash into pellets. This eliminates the danger of loss encountered to a greater or less degree by the best design of dry mash hoppers (even if no food be actually spilled, there is often a good deal carried on the beaks to the water containers) and the laboriousness of the wet mash method. Collection trays are often of glass, or may be of metal shaped somewhat in the form of a large funnel so that there is no loss of liquid when the trays are being washed. In experiments when laying hens are being studied, it is often of advantage to have some form of nest box which will allow excreta but not the eggs to fall on to the collection trays. As pointed out by Knowles, Watkin and Hendry [1933], care should be taken in mixing the mash to make sure that none of the mineral additions become sifted to the bottom of the container. If sufficient of the mash were to be used, the method adopted by Common would appear to eliminate a great many of the feeding problems in balance experiments with birds.

Many of the points mentioned above refer to balance experiments with fowls in general. Of possibly more particular interest in mineral balance studies with laying birds is to know how to treat the calcium and phosphorus of the egg. The time relationships for the passage of the developing egg down the oviduct, as given by Jull [1930], are as follows. The yolk is in the albumin secreting portion of the oviduct for about 3 hours, when most of the albumin is formed; it is in the isthmus for about one hour while the shell membranes are secreted and then goes to the uterus where the remainder of the albumin and part of the shell are secreted in 5–7 hours: the remainder of the shell is formed in the shell gland in 12–16 hours. Thus it may take from 21–27 hours for the egg to pass through the oviduct, and even then it may be "held" for some time. An egg laid early in the morning, then, should probably be credited to the previous day, but one laid late in the morning, or even in the afternoon must have been formed—provided there was not excessive delay in laying, after the egg was formed—partly the day before, and partly on the same day as it was laid. As most of the phosphorus of the egg is in the yolk, and in view of the time it takes for a yolk to become mature, the problem with phosphorus is further complicated. It has yet to be shown, also, how soon the "retained" calcium and phosphorus of the food is excreted by way of the egg. By totalling up the food and excreta calcium and phosphorus for more than one day, some of the inaccuracies due to these complications can be lessened; one may, however, be more interested in the daily mineral balance as affected by laying.

Halnan [1925] studied the N, Ca and P retention of two pullets. The periods he used were of 7 days duration, and the birds were on experiment for over 3 months. He fed a wet mash consisting of bran 2, Sussex ground oats 1, middlings 1 and fish meal 1, with the addition of 5·26% $CaCO_3$, and a grain ration of equal parts by weight of oats and wheat. Equal weights of grain and mash were fed. The diet of the birds contained 1·95% Ca and 1·26% P, a ratio of 1·55 : 1. He concluded that there was no early increased storage of Ca, but that the requirements for shell formation were met by increased retention during egg production. It appeared that before laying commenced the Ca was present in the excreta as $Ca_3(PO_4)_2$, the remaining food P being retained. During the egg laying periods, however, there was a heavy P excretion, which he thought was due possibly to the formation of egg fat from phosphatides [*cf.* Meigs, Blatherwick and Cary, 1919].

In their investigation on the utilisation of Ca from calcium carbonate and calcium citrate, Russell and McDonald [1929] used a basal ration of yellow maize 61, ground wheat 24, casein 12, NaCl 1 and cod-liver oil 2. $CaCO_3$ or Ca-citrate were added at the expense of the maize so that the ration had a Ca content of 1·4%. The ratio of Ca : P in the ration was 4·25 : 1, which was considerably wider than in Halnan's experiments. The authors confirmed Halnan's results with regard to the heavy excretion of P during egg laying, but, possibly due in part to the greater excess of Ca over P in the food, found more Ca in the excreta than was necessary to

combine with the P as $Ca_3(PO_4)_2$. They found no appreciable difference in the utilisation of the Ca supplied by the two salts.

Common [1932] made a somewhat more intensive study of the mineral balance of two pullets by doing daily analyses. His data indicated that with an adequate intake of Ca and P the retention of Ca increased as the bird was coming into egg production, there being apparently a preparatory storage of Ca and not, as Halnan concluded, a relatively short latent period between absorption from the food and its excretion as the egg shell. He studied the effects on the mineral balance of diets containing different levels of Ca and P. With a cereal ration having 0·067% Ca and 0·629% P (Ca : P = 0·11 : 1), or when 10% fish meal was added, thus increasing the food Ca to 0·697% and the P to 0·938% (Ca : P = 0·74 : 1), there was a higher percentage of P in the excreta than of Ca. About 2 weeks prior to the first egg being laid there was an increase in the percentage retention of Ca by the pullets on the fish meal diet, this being most noticeable in one bird which did not commence laying until the 30th day of the experiment.

In his second experiment the birds received U.V. Irradiation and a cereal ration containing approximately 7·3% soy bean meal as the protein supplement. For two birds this was supplemented with 3% $CaCO_3$, which resulted in the food containing 0·661% Ca and 0·451% P, a ratio of 1·47 : 1. These pullets throve very well. The Ca in the droppings fell to just under 0·2% while the percentage retention figures again indicated a preliminary storage of Ca in both birds about 20 days before the first egg was laid. During egg production Ca-retention (excluding that excreted in the egg, *i.e.*, Ca in food—Ca in droppings) was about 75%. During this time there was also an increased excretion of P in the droppings. With the same basal ration, but supplemented with 3% precipitated calcium phosphate, the food contained 0·455% Ca and 0·751% P, a ratio of 0·59 : 1. These birds also received U.V. Irradiation. One of the chief difficulties seems to have been that the birds were nervous and ate their eggs. While laying commenced early it was less regular and food consumption fell off quickly. (With regard to the nervousness Common cited Massengale [1929] who observed a similar condition in chickens receiving a Ca : P ratio in their diet of 0·33 : 1 but none when the ratio was 0·57 : 1). Notwithstanding the higher dietary P than in the $CaCO_3$ group, however, the percentage retention figures for Ca commenced to increase 10–14 days before the first egg was laid, and there was the same tendency for the minimal Ca excretion to be about 0·2 g. daily. Common pointed out that one would gather from Halnan's data that there was an obligatory excretion of over 1 g. Ca daily in the droppings. This was higher than that observed by either the American workers or Common, but the latter suggested that Halnan's birds were possibly suffering from a lack of vitamin D and their fæcal excretion of Ca might have been high as a result. As was mentioned previously, where no antirachitic factor was supplied to chickens, the Ca content of the ration had to be high to get at all good growth. So Common concluded from his data and those of Russell

and McDonald, that when vitamin D was supplied there was an obligatory excretion of the order of 0·18 g. Ca in the droppings.

In his second paper Common [1933] substantiated the conclusions he had reached with regard to the increased retention of calcium and phosphorus preparatory to egg laying, and concluded from the Ca : P ratios of the " retention " that these minerals were probably being deposited as bone salts. He quoted the results of Halnan, together with his own, and showed that the ratio was in the neighbourhood of 1·21 : 1 [$CaO : P_2O_5$]. He also presented further data illustrating the heavy excretion of phosphorus which occurred prior to the laying of an egg. This excretion amounted in some cases to as much as 2–2·5 times the P intake, although before laying commenced the P-balance was positive. In consideration of the time it takes for an egg to be formed and that as a rule the increased excretion occurred not later than the same day an egg was laid, he concluded that there was a close connection between shell formation and the heavy P-excretion. His view is that during the pre-laying stage, Ca is stored in the bones (probably as calcium phosphate), which accounts for the increased Ca-retention. During laying the shell-Ca is derived from that in the food, if conditions be such that normal absorption and assimilation can proceed. If, however, for certain reasons—such as insufficient vitamin D or Ca—normal metabolism cannot continue, the bony stores are drawn upon to make up the deficit. The Ca in the bones being (as is generally supposed) largely in combination with P, release of Ca results in release of P as well. This accounts for the increased P excretion which occurs about the time the shell is being formed. The Ca : P ratio of these elements " retained " during the pre-laying period was taken as confirmatory evidence for his thesis.

With regard to the combination in which Ca and P occur in the excreta, it has been mentioned that Halnan considered that during non-laying the actual values for excreta-Ca agreed with that required by theory to combine as $Ca_3(PO_4)_2$ with all the P in the droppings. The additional food-P had therefore been retained. During the laying periods the P-excretion was in excess of that required by theory to combine with the Ca in the excreta. Russell and McDonald were unable to confirm this, there being too little P excreted during the non-laying period for all the Ca to have occurred in the droppings as tricalcium phosphate. Common's data, also, do not agree with the theoretical requirements. Knowles, Watkin and Hendry [1933] estimated the carbonate as well as the Ca and P content of the excreta and concluded that there was a greater probability of the P being present as dicalcium phosphate, the remaining Ca being as the carbonate. This, from their data, would seem irrespective of the carbonate content of the ration, within the limits they used. In the three diets they fed, the total calcium was from 2–3%, the total phosphorus about 1·5% and the calcium carbonate from 0·89–5·57%. The Ca : P ratios ranged from 1·47–1·90 : 1. Their pullets, with the exception of one which was ovulating intra-abdominally, were not laying, and apparently not coming into lay. In Common's experi-

ments it is evident that during egg laying all the P could not have been excreted as dicalcium phosphate, and in only a few instances before laying commenced could it have occurred in that form in the droppings. Calculations show, indeed, that during a large part of the experiments there was insufficient Ca in the excreta to combine with all the P as even the monocalcium salt. It would appear, then, that P need not necessarily occur in the excreta in Ca-combination alone. Common thought the extra phosphorus was excreted in the urine probably, as he found the water-soluble phosphates on these days to be high.

Common's analyses included determinations of Na and K. Two pullets received a cereal ration containing 0·94% CaO and 0·93% P_2O_5 while two others received the same ration with the addition of 0·5% NaCl. He concluded that Ca retention was considerably better in the NaCl supplemented ration, this being an effect of the Na rather than the Cl ion.

It is known that during egg laying the serum-Ca of hens increases to very high values, and MacOwan [1932] observed a certain relationship between the serum-Ca and the weight of the largest egg in the oviduct. While her data did not indicate exactly in what part of the oviduct this egg was found, she concluded that the peak in the serum-Ca occurred just prior to shell formation, and that it fell as the shell was being formed, reaching its lowest value when the shell was fully formed. Charles and Hogben [1933] were unable to confirm this. They studied the levels of serum-Ca and Mg of pullets in relation to the position of the egg in the oviduct, and found that no close connection existed. Their results were as follows :—

1. Sexually immature pullets :—Serum-Ca 9·9–12·2 mg. ; serum-Mg 2·08–2·71 mg./100 ml. serum.
2. Laying pullets, no egg in oviduct :—Serum-Ca 15·0-19·6 mg. ; serum-Mg 1·87–2·87 mg./100 ml. serum.
3. Laying pullets, eggs in albumin or shell membrane secreting portions :—values similar to (2).
4. Laying pullets, eggs in various stages of shell-formation :—Serum-Ca 10·4–28·5 mg. ; serum-Mg 1·86–5·90 mg./100 ml. serum.

These authors considered that the hypercalcæmia was a result of the functionally active ovary, and that Ca-excretion as egg shell being in advance of mobilisation would account for the cases which showed no increased level of serum-Ca. Among other reports dealing with the subject might be mentioned that of Buckner, Martin and Insko [1930] who found that there was approximately 12% more Ca in blood from the anterior mesenteric vein than in the anterior mesenteric artery.

A study of the forms in which Ca exists in the serum of hens has shown that during hypercalcæmia there is no increase in the ultra-filtrable fraction : the increase is all in the non-ultrafiltrable [Laskowski, 1933 ; Correll and Hughes, 1933 ; Benjamin and Hess, 1933]. Some of Laskowski's results

follow. The increase in non-filtrable-P he considered evidence for the formation of a Ca-P complex.

Condition.	Plasma-calcium.			Plasma inorganic-P.		
	Total (mg./100 ml.).	Ultra-filtrable (mg./100 ml.).	% of total.	Total (mg./100 ml.).	Ultra-filtrable (mg./100 ml.).	% of total.
Non-laying (5 birds)	12·7 (11·8–13·0)	8·0 (7·1–9·3)	65 (55–77)	2·9 (2·0–4·6)	2·8 (2·0–3·7)	97 (83–114)
Laying (7 birds) ...	20·7 (16·3–25·8)	8·0 (7·4–8·6)	39 (29–49)	4·1 (2·3–5·6)	2·2 (1·3–3·9)	54 (39–70)

Similar values for the serum-Ca were obtained by Correll and Hughes. Benjamin and Hess determined, in addition, the amount of the total and filtrable-Ca which was adsorbed on $BaSO_4$. Thus they had four fractions :—

A. Non-filtrable (1) adsorbable (Ca-P complex).
(2) non-adsorbable (protein bound).
B. Filtrable (3) adsorbable (Ca-P complex).
(4) non-adsorbable (remainder or " ionic ").

During artificial hypercalcæmias in dogs produced by parathormone, vitamin D or the injection of Ca-salts, there was an increase in all four fractions, but chiefly in the filtrable. In the normal hypercalcæmia of hens, on the other hand, the increase was in the two non-filtrable fractions only. It has yet to be shown whether artificially produced hypercalcæmia in hens will give similar results or whether the response will be the same as it is in dogs.

Schmidt [1932, 1] determined the retention of Ca, P and N by four young fowls (535–1,000 g. in weight), two laying hens (2,035–2,270 g.) and four non-laying hens (1,255–2,085 g.). These were fed basal diets containing either flaked potatoes (physiologically slightly alkaline) or cereals (physiologically slightly acid) together with protein supplements and a vitamin D preparation. The diets were fed alone and with additions of $CaCO_3$ or $CaCl_2$. The conclusion reached was that with the potato diet there was better retention of Ca with $CaCl_2$ than with $CaCO_3$, while with the cereal ration the reverse was the case. Ca was better retained when the ration was physiologically neutral or slightly acid ; strongly acid or alkaline diets were adverse to Ca-retention. The influence of the dietary Ca : P ratio on the proportion of Ca and P retained was only noticeable when the diets were physiologically neutral or slightly acid. The data were not entirely clear cut, for in some cases there was somewhat better retention of Ca and P when the chloride was added to the cereal ration than when the carbonate was

added, and similarly when the carbonate was added to the potato diet. As a practical test of his conclusions, however, the author fed 40 hens on a cereal-fish meal ration and noted the egg production. The addition of 50 g. moist potato per bird daily to make the ration physiologically neutral, resulted in an improvement in egg production without the addition of more mineral supplement. Whether the improvement noted was due entirely to the action of the potatoes in adjusting the physiological acid-base equilibrium of the diet or to some other cause also, would seem to require further elucidation.

Hatchability.

A considerable amount of money is lost annually through poor hatchability of eggs placed in incubators. The loss is probably greater than formerly, owing partly, but not necessarily, to intensive methods practised at present and to the tendency for earlier hatching of chicks. Many of the factors concerned have been studied. Fertility of the eggs is of primary importance. Recent work has demonstrated that this is affected, among other things, by the age of the bird, eggs of old hens being as a rule less fertile than those of pullets or yearlings [*vide* Greenwood, 1931–32]. Some individual hens tend to lay a higher percentage of fertile eggs than others, and Hindhaugh [1931–32] suggested that after the first test it would ordinarily be advisable to discard for breeders those hens showing low fertility, as it was probable they would continue to do so during the hatching season. Jull [1927–28, *et seq.*] found that intercrossing of inbred strains of the same breed resulted in a lowered fertility of the eggs. Card [1928–29] found that when pullets were deprived of vitamin E they laid infertile eggs, but when the vitamin was supplied by wheat germ oil the fertility improved. The addition of wheat germ oil did not appear to increase the fertility of birds fed a good "practical" ration. Excessive fatness of either the males or females has also been suggested as a factor causing low fertility.

Recent work, on the basis of hatchability of fertile eggs set, has included a study of the effect of genetical, nutritional and environmental factors.

Jull's investigations [1927–28, *et seq.*] have shown that inbreeding without careful selection will lower the percentage hatchability; continuous full brother-sister mating is worse than full brother-sister mating alternated with half brother-sister mating. The decrease in hatchability was caused by an increase in embryonic mortality, especially during the 18th to 21st days of incubation. The greatest effect was during the first year's breeding but there was also a general decline of fertility in successive years. The same coefficients of inbreeding gave approximately the same percentage hatchability, irrespective of the year the hatch was tested. Careful selection on the basis of hatchability records, etc., should result in improvement of hatchability. It has been shown that breeding and feeding for high egg

production need not necessarily result in decrease in the hatching power of the eggs [*vide infra*].

Nutritional factors affecting hatchability have received considerable attention recently. The influence of certain animal protein supplements and of vitamin D has been studied at the Ontario Agricultural College. The basal mash consisted of yellow maize, oats, wheat shorts, alfalfa meal, bone meal and common salt. The grain mixture consisted of yellow maize and wheat. No artificial lighting was used and the birds, in groups of 20, were confined to the pens for eleven months of each year of the experiments. Sun could enter the pens on fine days. Late March to early April-hatched pullets were used as it was considered these would give poorer hatchability than yearling hens. Various animal protein supplements were fed, these being given singly in such amounts as to bring the crude protein of the mash to 21% (except when buttermilk powder was used), or in combination with buttermilk powder to raise the crude protein of the mash to approximately 18%. Cod-liver oil was given with some of the rations and U.V. Irradiation to one group of pullets receiving 10% buttermilk powder. The experiments have been carried on for over five years, the birds being put on experiment in October of each year and eggs set daily in a 6,000 egg Petersime incubator from February to July inclusive. The following summary is taken from the report on the results for the first three years. [Graham 1930 ; Graham, Smith and McFarlane, 1931] The paper by Smith [1933] contains a detailed account of the distribution of embryonic mortality.

The average hours of sunshine were as follows :—

Spring months.	389·8 hours.	Summer months.	823·1 hours.
February	100·2 hours	May	246·1 hours
March	126·2 ,,	June	263·8 ,,
April	163·4 ,,	July	313·2 ,,

With the various protein supplements, fed singly and without cod-liver oil, the average hatchability during the spring months was of the order : 10% milk (57%) ; fish scrap (56%) ; beef scrap (47%) ; tankage (20%). During the three summer months the hatch improved to : 10% milk (73%) ; fish scrap (66%) ; beef scrap (26%) ; tankage (46%). When cod-liver oil was added to the rations the hatch improved 11–18% in the spring months, except when fish scrap was used. This brand of fish scrap was found to possess sufficient antirachitic value for growing chickens, without the addition of cod-liver oil [McFarlane, Graham and Richardson, 1931]. During the summer months cod-liver oil did not improve the hatchability of the eggs, except possibly when beef scrap was used. A combination of buttermilk powder and fish scrap did not result in a higher hatch than when fish scrap

alone was used as the supplement. When buttermilk powder was used to supplement rations containing cod-liver oil with fish scrap, beef scrap or tankage, however, the hatchability was improved considerably in the spring. The best results were obtained during these months (Feb.–Apr.) with a combination of buttermilk powder and cod-liver oil with either fish scrap or beef scrap (approximately 71% hatchability). Heavy egg production in itself did not appear to depress hatchability. Tankage fed alone or in combination with milk and cod-liver oil did not give such good results as did the other supplements. Under the conditions of the experiment it appeared that until there were over 200 hours of sunshine per month, the birds did not receive sufficient vitamin D unless cod-liver oil or ultra-violet irradiation were given. Under less closely confined conditions which may prevail in places having a more temperate winter climate, it is probable that birds will receive sufficient sunshine when there are less than 200 hours of sunshine per month.

Cod-liver oil would seem to be more efficient as a vitamin D supplement for laying birds than irradiated ergosterol preparations. In the experiment reported by Branion and Smith [1932], pullets were kept indoors in batteries and given the ration mentioned previously [Graham, *et al.*, 1931], containing 10% buttermilk powder. The average percentage hatchability of fertile eggs laid by the six groups of pullets for six months was as follows.

Ration	Per cent Hatchability	
Basal (control)	16·7	
2% cod liver oil	49·2	
2% 10 D cod liver oil	43·6	(cod-liver oil fortified with viosterol).
2% diluted viosterol	23·6	(viosterol diluted to equivalent rat units of cod-liver oil).
2% 250 D viosterol	68·3	
10,000 D viosterol	13·2	(1 ml. per hen daily, *per os*).

Note:—D is used to signify the antirachitic potency for rats of "standard" cod-liver oil, the preceding number indicating the potency of the preparation in terms of the "standard"; e.g., a solution 250 D is 250 times as potent for rats as the same quantity of "standard" cod-liver oil. Viosterol is an irradiated ergosterol preparation.

The amount of vitamin D supplied by viosterol necessary to give an effect equivalent to that of 2% cod-liver oil, was just under 250 D (or in view of the critique by Bills, *et al.* [1931], at least 100 times the rat units supplied by 2% of "standard" cod-liver oil). The pullets in the last group received 1 ml. 10,000 D viosterol orally each day, or 1,000,000 International units (if D = 100 International units). At the beginning of February the hatchability of their fertile eggs was over 50%, but fell rapidly to about 15% by March, to about 2% by April 1, and ceased to hatch after the end of

April. Egg laying did not cease entirely but there was considerable loss of body weight and egg weight. The incidence of monsters of the ectopic and chondrodystrophic types increased. Embryonic mortality occurred chiefly during the second week of incubation [Smith, 1933].

In their study of the effect of the diet on egg composition Titus and his collaborators [Titus, *et al.*, 1933, 1 ; Byerly, *et al.*, 1933, 1, 2 ; Ellis, *et al.*, 1933] found that when the dietary proteins were from cereal sources alone, the embryonic mortality of the eggs produced was high, especially during the second week of incubation. Supplementing the diets with certain animal proteins corrected this high second week mortality. Menhaden fish meal (flame-dried) and certain lots of meat meal, however, were little better than vegetable protein supplements, so that it appeared that some process in their manufacture was possibly at fault. The supplements found to result in " normal " embryonic mortality were vacuum-dried N. Atlantic fish meal, crab meal (steam-dried), buttermilk powder or a combination of meat and fish meal and buttermilk powder. When the birds had access to blue grass range they appeared able to correct deficiencies in their diet : range had, however, little supplemental value for the " normal " ration, from the standpoint of hatchability of the eggs produced.

All the rations were supplemented with minerals and cod-liver oil and the birds had access to " sun parlours " when they were confined, so that it would not seem there was a deficiency of the common minerals or of vitamin D. Eggs produced by hens receiving the well-balanced diet containing meat and fish meal had a greater weight of dry matter in both yolks and whites than when the dietary protein was from vegetable sources alone ; with crab meal, the eggs contained a higher percentage of protein in the yolks than either the eggs produced on the basal or the well balanced rations [Titus, Byerly and Ellis, 1933]. McFarlane, Fulmer and Jukes [1930] could not find any significant differences in their analyses of the amino-acid N distribution in eggs produced by hens receiving diets supplemented with tankage (very low hatchability) or buttermilk powder (good hatchability).

Ellis, Miller, Titus and Byerly [1933] found that with diets low in the vitamin B complex there was from one-third to one-half as much vitamin B_1 in the eggs as when there was an adequate supply of vitamin B. The vitamin B_2 content was not affected to the same degree. Nevertheless the differences in the vitamin content of the eggs seemed unrelated to the hatchability of the eggs. It has been suggested [Macdonald, *personal communication*] that if birds be limited to a ration which has been exposed to great heat [such as may occur in the manufacture of pellets] they often suffer from what appears to be a real or relative vitamin B deficiency. Pellet feeding to hens sometimes results in very low percentage hatchability of the eggs, and yeast seems to act as a corrective. To which factor (if any) of the vitamin B complex this is due is not clear at present.

There was a high embryonic mortality in eggs produced by pullets receiving tankage as the protein supplement, in the experiments at the

Ontario Agricultural College. This was related in part to the incidence of anæmic-like embryos which died during the early part of incubation. Analyses of eggs produced by pullets on the tankage diet did not suggest that there was a greater deficiency of iron or of copper than there was in eggs produced by birds on the other diets [McFarlane, Fulmer and Jukes, 1930]. Analyses of the blood of hens producing such embryos, by Cavers and Hutt [1933], did not show abnormal values for number of leucocytes or erythrocytes or percentage of hæmoglobin. Bronkhorst and Hall [1933] did not find any difference between high and low hatching hens with respect to number of erythrocytes or percentage of blood hæmoglobin, glucose and phosphorus.

Summary.—From the experiments reviewed in this section the conclusion might be drawn that under conditions of confinement, birds kept for breeding probably require an animal protein supplement in their ration. There is considerable difference in the value of these : good grades of milk powders, fish meal and meat meal are probably better than tankage and those products which are treated to high temperatures in their manufacture. A mixture of supplements would seem to be an improvement upon any one alone, if there be no other limiting factors. It has to be remembered, of course, that in some of the experiments it is not necessarily the effect of the protein of the supplement, *qua* protein, which results in improvement of the hatchability of the eggs, but that one product may possess certain other qualities which are limiting factors in another supplement. When birds have access to good range they appear able to make good many deficiencies connected with the type of protein supplement used.

Vitamin D deficiency is an important factor in causing poor hatchability, and can probably be supplied most efficiently at present, for birds in confinement, by fish-liver oils. The need for additional vitamin D is correspondingly less with increase in the amount and quality of sunshine available for the birds.

Of the environmental factors, good grass range is of no small importance in allowing the birds to supplement their dietary defects. This is probably one of the reasons why it is only fairly recently that large scale experiments have been carried out in Great Britain on the effect of nutrition on hatchability.

The time an egg is laid may be a possible factor also. Hutt and Pilkey [1929–30] found that eggs laid before 9 a.m. hatched better than those laid after 12 noon, especially during the early part of the hatching season. They suggested that this might be related to chilling of the eggs at a critical stage in the development of the embryos, which stage had been passed by the embryos in eggs laid before 9 a.m. Nicolaides [1933] was unable to confirm the difference in hatchability between eggs laid before 9 a.m. and after 12 noon, but found from cytological studies that embryos in newly laid eggs from high hatching hens were further advanced in development than those in eggs from low hatching hens, regardless of the time of laying. Funk

[1933] found that eggs laid in the afternoon hatched better than those laid in the morning, and during an extremely cold week there was a significant decrease in the hatch from eggs laid before 9 a.m.

Hale [1929–30] suggested that embryonic malposition might be related in part to incorrect incubation, the turning of the eggs and the angle at which they were placed in the incubator being important. The extensive work on the numerous malpositions and malformations of embryos is not reviewed here. Indications at present point to the effect of genetical and nutritional as well as environmental factors in preventing these abnormalities.

General Summary.

In this monograph are discussed some of the experiments dealing with six phases of poultry nutrition, *viz.*, " purified " diets ; the vitamins ; the minerals ; " leg weakness " ; mineral balance experiments and hatchability.

It appears possible to rear chickens to maturity with a fair degree of success on " purified " diets but it is as yet too early for one to say what are their exact nutritional requirements. Of the vitamins, A, D and E appear most easily supplied in adequate amounts. At present it is not possible to state all the factors of the vitamin B complex which are necessary : vitamins B_1 and B_2 are most certainly required, B_4 very probably and B_3 possibly. The other factors of the complex necessary for normal growth have not yet been classified. Work on those minerals which are most likely to be present in sub-optimal or excessive amounts has been discussed, and the effect of these levels on the health of growing birds dealt with. Calcium and phosphorus metabolism is affected by the form in which they are fed as well as by the presence of vitamin D and of other elements in the ration. Discussion of " leg weakness " has taken place under the headings of those forms caused by micro-organisms and those more intimately related to errors in the diet. The chief symptoms typical of the conditions have been outlined, together with what appear at present to be curative and preventive measures. The few experiments on record dealing with the retention of calcium and phosphorus have been reviewed. It would appear that a further study by means of balance experiments of the best forms in which to feed these elements, and of the importance of vitamin D and of other factors on their proper utilisation should prove of value in the correct management of the flock under intensive conditions. The proper development of chicken embryos is affected by many things, not the least of which is the nutrition of the parents. In this connection factors of primary importance are the supply of vitamin D and the quantity and quality of the protein supplements. Analyses of eggs of high and low hatchability have not shown that the amino-acid N distribution is significantly different in the two cases, and it is not unlikely that some of the B vitamins are limiting factors.

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By Harrison and Sons, Ltd., 44-47, St. Martin's Lane, W.C.2.

(B 12/4932)x Wt. P. 1623-60 750 11/34 H & S, Ltd. Gp. 12